THE PLANT DISEASE REPORTER

CROPS RESEARCH DIVISION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

U. S. DEPT. OF AGRICULTURE
LIBRARY
DEC 12 1961
CURRENT SERIAL RECORDS

Volume 45 Number 11

November 15, 1961

Growth Through Agricultural Progress

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

SUGGESTIONS FOR PREPARATION OF MANUSCRIPTS

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

(2) MANUSCRIPTS should be the original ribbon copy, not carbons, clearly typed and double-spaced throughout, including tables, footnotes, and literature cited. Submission implies nonsubmission elsewhere and (if accepted) no publication elsewhere in the same form without consent.

(3) ABSTRACTS are requested for all except very short articles.

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so.

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. Place the year of publication after the author's name. Please check your references carefully since we cannot always do it for you. Be sure that text citations and bibliography agree. Cite number or month for periodicals that are not paged consecutively throughout the volume.

(6) PESTICIDES must be listed first by their chemical names. If the chemical has an approved common name it must follow, in parentheses. Where a common name is available, use of a trade or proprietary name is optional and, if given, should be listed last, in parentheses.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

(8) TABLES should be carefully thought out with particular attention to the Reporter's limitations in reproduction. Make titles and headings definite and self-explanatory. Designate footnotes in tables with superscript lower-case letters. Be sure that text discussion agrees with the data in the table. Do not abbreviate names of crop varieties.

(9) REPRINTS cannot be supplied since there is no way in which we can be reimbursed. However, the MULTILITH PLATES from which reprints can be made will be sent if requested at the time the article is submitted. If reprints are desired of articles other than your own, please request them direct from the authors.

ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to twelve double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases.

Paul R. Miller

Manuscripts for and correspondence about this publication
should be sent to:

PLANT DISEASE REPORTER
Epidemiology Investigations, Crops Protection Research Branch
Plant Industry Station, Beltsville, Maryland

THE PLANT DISEASE REPORTER

Crops Research Division, Plant Industry Station, Beltsville, Maryland
Volume 45 November 15, 1961 Number 11

CONTENTS

1. A staining-fungus root disease of pondersoa, Jeffrey, and pinyon pines
WILLIS W. WAGENER and JAMES L. MIELKE 831

2. Paxillus atrotomentosus causes brown root rot in dead jack pine in plantations in Wisconsin
ROSS W. DAVIDSON and ROBERT F. PATTON 836

3. Studies on the occurrence and distribution of the oak wilt fungus in oak leaves
H. C. THOMPSON 839

4. Three uncommon watermelon fruit rots in Florida
N. C. SCHENCK 841

5. Observations on new or unusual diseases of highbush blueberry
L. C. RANIERE 844

6. Fusarium solani f. pisi, a cause of pea root rot in New Hampshire
GRAYDON C. KINGSLAND and AVERY E. RICH 845

7. Resistance in Triticum vulgare to infection by Erysiphe graminis f. sp. tritici as influenced by the stage of development of the host plant
L. W. BRIGGLE and A. L. SCHAREN 846

8. Distribution of some cucurbit viruses in some Southwest melon-producing areas
R. E. WEBB 851

9. An outbreak of Cronartium comptoniae on Monterey and Bishop pines on Vancouver Island, British Columbia
A. C. MOLNAR 854

10. Spread and distribution of camellia flower blight in Louisiana
D. M. ALFORD, et al. 856

11. Control of Pythium root diseases with soil fungicides
J. TAMMEN, et al. 858

12. Control of Pythium root and basal stem rot of Chrysanthemum morifolium with Dexon
J. TAMMEN and D. P. MUSE 863

13. A non-fungistatic bacteriostatic medium containing streptomycin and vancomycin
A. J. LATHAM and M. B. LINN 866

14. Trunk canker of macadamia in Hawaii caused by Phytophthora cinnamomi Rands
RICHARD B. HINE 868

15. Turfgrass diseases in southern California
R. M. ENDO 869

16. The relation of plant fertility to bacterial spot of pepper
W. W. TOWNSLEY and D. F. CROSSAN 874

17. Aphis gossypii, a vector of citrus vein-enation virus
E. F. LAIRD, Jr. and L. G. WEATHERS 877

18. Sources of resistance to the cowpea yellow mosaic virus
DARRELL G. WELLS and RAPHAEL DEBA 878

19. Occurrence of viruses causing raspberry mosaic in some commercial stocks of red raspberry in eastern United States
RICHARD H. CONVERSE 882

20. Soil-borne mosaic of fall-seeded oats in Western Washington
G. W. BRUEHL and V. D. DAMSTEEGT 884

21. Pinus radiata susceptible to pitch canker
GEORGE H. HEPTING 889

22. Some evidence that pear bark measles in seed-borne
C. B. CORDY and I. C. MacSWAN. 891

23. The effect of pesticides on turfgrass disease incidence
JOHN H. MADISON 892

24. Cereal rust epidemiology in Kansas in 1959
L. E. BROWDER, et al. 894

25. Juncus effusus, a new host for Epichloë typhina
R. A. KILPATRICK, et al. 899

26. Dutch elm disease in Arkansas
CHARLES L. WILSON, et al. 900

27. Hoja blanca in El Salvador
OSCAR ANCALMO and GUILLERMO RAMOS SILIEZAR 900

28. A severe epidemic of Helminthosporium brown spot disease on cultivated wild rice in northern Minnesota
GEORGE A. BEAN and RICHARD SCHWARTZ 901

A STAINING-FUNGUS ROOT DISEASE OF PONDEROSA, JEFFREY, AND PINYON PINES

Willis W. Wagener and James L. Mielke[1]

Summary

A killing root disease of ponderosa, Jeffrey, and singleleaf pinyon pines in parts of California also occurs on pinyon pine in western Colorado and northeastern Arizona. It is similar to, if not the same as, one reported on planted eastern white pine in Montana and is caused by a staining fungus, Verticicladiella sp. No perfect stage has been found. Unlike the usual sapstain fungi, which concentrate chiefly in the medullary rays forming wedge-shaped patterns in the sapwood, this fungus grows through the wood tracheids, without a wedge-shaped pattern. Local spread of the fungus from tree to tree is by contact of diseased and sound roots. Contact infection of roots has been demonstrated experimentally. Root systems of individual trees are progressively invaded, stained, and killed, with accompanying reductions in tree growth until the tree dies. Bark beetles sometimes invade mortally affected trees. In pinyon stands the disease progresses at a rate of about 40 feet in 6 years. Mortality from it fluctuates, apparently influenced by soil moisture. The fungus seems to represent a new type in forest tree pathogens.

A root and butt disease, producing a dark stain in affected wood and causing progressive mortality in plantations of eastern white pine in Montana, was recently reported by Leaphart[2]. The evidence indicated that the pathogen responsible was a staining fungus, Verticicladiella sp.

A similar disease has been known for many years in native ponderosa (Pinus ponderosa), Jeffrey (P. jeffreyi), and singleleaf pinyon (P. monophylla) pines in California, and in pinyon pine (P. edulis) in Colorado and northeastern Arizona.

HISTORY

The disease in California was first found in 1938 by forest entomologists[3] in the course of insect investigations on Blacks Mountain Experimental Forest, Lassen County, California. An immature ponderosa pine showed incipient top fading without the presence of bark beetles in the trunk below. Purplish-brown streaks of stain were found in the outer sapwood at the base of the tree and these were later found to connect with more pronounced stain in the roots. Localized foci of the disease were subsequently found in ponderosa and Jeffrey pines in several other compartments of the Experimental Forest and in the Willow Creek drainage, western Modoc National Forest.

In 1941, forest entomologists J. M. Miller and D. DeLeon discovered a similar appearing stain in the bases and roots of dying singleleaf pinyon pines in a basin adjoining Baldwin Lake, San Bernardino National Forest, in California. The disease was found to be quite general in singleleaf pinyon stands in the area (Fig. 1).

In 1942, the junior author and S. T. Carlson of the National Park Service discovered the disease in pinyon pine in Mesa Verde National Park, Colorado. It was later found to be common over a large area in the Park and to extend for about 40 miles north and east of the Park boundary. A localized infection center was also located in pinyon pine north of Newcastle, Colorado. In 1945, it was noted as occasional in pinyon in Canyon de Chelly National Monument in northeastern Arizona[4]. In 1957, pathologists J. W. Kimmey and H. H. Bynum discovered a localized infection in ponderosa pine northeast of Jackson, Amador County, California. This is the only case of the disease that has been found on the west slope of the Sierra Nevada.

---

[1]Respectively Plant Pathologist, Pacific Southwest Forest and Range Experiment Station, Berkeley, California and Plant Pathologist, Intermountain Forest and Range Experiment Station, Ogden, Utah. The observations and tests reported were made under the former Bureau of Plant Industry, Soils, and Agricultural Engineering, United States Department of Agriculture. Pathologist J. W. Kimmey participated in field work on the disease in California. Jack J. Wade, Chief Ranger, National Park Service, Mesa Verde National Park, Colorado, rendered valuable field assistance. Marion S. Cave, Doris A. Hardman, and Bessie Etter assisted in laboratory phases.

[2]Leaphart, Charles D. 1960. A root stain disease of eastern white pine. Plant Disease Reptr. 44: 704-706.

[3]Reported, with specimens, by K. A. Salman.

[4]Letter, June 28, 1945, Harold M. Ratcliff, Forest, National Park Service, Santa Fe, New Mexico.

Field evidence indicates that the disease is of long standing in Mesa Verde National Park, Colorado and in the Baldwin Lake area in California. It is probably native in all places where it has been found.

During 1933 and 1934, about 12,000 dead and dying pinyon pines were cut and burned within Mesa Verde National Park by work crews for cleanup purposes. Representative stumps from this cutting were sampled 10 years later and a large proportion were found to contain the stain typical for the disease. From what is now known, the disease was undoubtedly the chief primary cause of the death of pinyons in the Park between 1932 and 1934.

SITE CONDITIONS

For the most part the disease occurs in districts with a relatively low annual precipitation, 18 to 20 inches in northeastern California and western Colorado and 30 inches but very variable in southern California. Precipitation averages about 55 inches in the limited center in Amador County, California, but summers are dry. Elevations in infected districts are 4000 feet in Amador County, 5600 to 5800 feet in northeastern California, 6700 to 7000 feet near Baldwin Lake, California, and 6200 to 8000 feet in western Colorado.

Representative mean monthly minimum temperatures are 1.5° F at Blacks Mountain and 15° at Baldwin Lake in California; 18° at Mesa Verde National Park in Colorado. Corresponding mean monthly maximums are 85°, 77°, and 83°. Soils are rather fine textured but friable in most of the localities, but a light and very rocky loam near Baldwin Lake. At Mesa Verde the incidence of the disease is highest in the deeper soils toward the centers of the mesas.

SYMPTOMS

Manifestations of the disease in ponderosa, Jeffrey, and pinyon pines are quite similar to those described by Leaphart[2] in Montana plantations of eastern white pine. Affected trees slow down in growth for 1 to 8 years before death, depending on their size and on the progress of the fungus through the root system. For the year in which dying occurs the trees make no growth. Bark beetles or borers often invade the trees during this final stage.

As a representative case history in ponderosa pine in California an off-green color was noted in a 40-inch diameter tree in June[5]. No bark beetle infestation was present. In mid-August a dark stain was found in the basal sapwood. Early in September the trunk was attacked by western pine beetles (Dendroctonus brevicomis Lec.). By that time stain streaks extended up in the outer sapwood for several feet above ground level, the longest to 7 feet, and to depths of 0.3 to 3.0 inches from the cambium. The same stain was present in the roots and in parts was accompanied by heavy resin infiltration. Some stained roots had been dead for several years. Radial growth of the tree had progressively declined for 7 years.

Within the zone of root spread of the tree, three young Jeffrey and two ponderosa pines 2.5 to 6.0 feet high were either dead (two) or showed impaired growth for the current year. All had the same stain in the main roots as was present in the roots of the large tree.

In pinyon and singleleaf pinyon pines the stain seldom extends for more than a foot or two above ground level (Fig. 2). In all species its color is variable, depending on the moisture content of the wood, on whether the stained wood is resin-infiltrated, and on whether the cambium is still alive. The color may range from an intense black through dull or grayish black to brown or purplish brown. The actual color of the individual hyphae in the wood is an orange-brown.

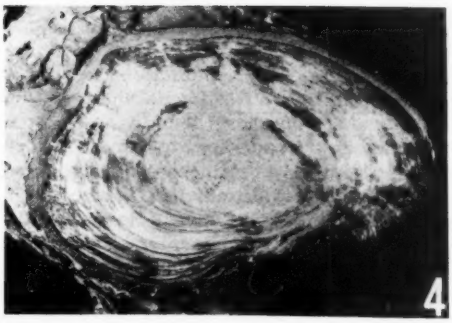
A particular feature of the stain pattern in the sapwood is that it is not wedge-shaped, as in most sapstains, but tends to follow the annual rings (Figs. 3 and 4). Most sapstain fungi concentrate in the medullary rays and advance along them, whereas the root-stain fungus progresses almost entirely through the tracheids and does not concentrate in the rays (Fig. 5).

Affected pinyons are sometimes invaded before their death by Ips beetles. Broods originating in such trees sometimes attack and kill neighboring pinyons to which the disease has not extended. This happened on four plots established to follow the progress of the disease, but the percentage of pinyons killed directly by Ips attacks did not on any plot exceed 9% of the numbers killed primarily by the root disease. On all plots, losses from primary Ips attack were confined to 1 to 2 years out of the 7 or 8 that the plots were under observation.

In ponderosa and Jeffrey pines the disease is probably more common than discovery records to date indicate because symptoms of its presence are frequently obscured in the final stages by insect attacks.

---

[5]Observations in part by forest entomologist J. W. Bongberg.



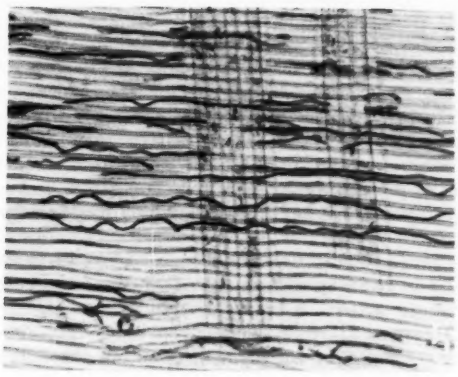


FIGURE 1. Killing of singleleaf pinyon by Verticicladiella sp., Baldwin Lake, San Bernardino National Forest, California. In addition to the killed group in the left foreground, killed groups appear in the middle distance.

FIGURE 2. Stain from the root disease in the butt of a singleleaf pinyon about 5 inches in stem diameter at the cross-section cut.

FIGURE 3. Cross section of the basal stem of a small singleleaf pinyon dying from the root disease, showing stain in the sapwood. Note the absence of a wedge-shaped pattern in the stain.

FIGURE 4. Stain from the disease in a pinyon pine root, Mesa Verde National Park, Colorado. National Park Service photo.

FIGURE 5. Sinuate hyphae of the pathogen in the basal sapwood of ponderosa pine. Note the absence of hyphae in the medullary rays. Longitudinal section.

THE INDUCING FUNGUS

The first successful isolation of a fungus corresponding to that found in the stained wood was made by the late Caroline T. Rumbold at the Forest Products Laboratory, Madison, Wisconsin. The isolation was from stained chips from ponderosa pine. Miss Rumbold provisionally identified the isolate as Leptographium lundbergii Lag. & Melin, but later study of other isolates from ponderosa and pinyon pines by Marion S. Cave demonstrated that the fungus was not L. lundbergii. It seemed to be closest to the Leptographium stage of Ceratocystis (Grosmannia) serpens (Goid.) C. Moreau, although not that species. Our fungus has recently been assigned to the genus Verticicladiella[6]. No species determination has yet been made. In culture, ellipsoid hyaline conidia are produced on the penicillately branched heads of stout conidiophores of the Leptographium type, arising from the mycelium. No perfect stage of the fungus has been found.

In invaded sapwood the hyphae follow a sinuous, lengthwise course through the tracheids (Fig. 5), passing from tracheid to tracheid through the bordered pits. They are almost never found in the medullary rays. Fully developed hyphae in wood average about 5.6μ in width.

Difficulty has often been experienced in attempting to isolate the fungus from infected sapwood or root wood but this may have been because most isolations were attempted at room temperature. A recent discovery by pathologist R. V. Bega[7] of this Station that an isolate from singleleaf pinyon had a growth optimum at about 15°C and that at usual room temperatures it grew quite slowly suggests a reason for some of the failures.

In California there appear to be two strains of the fungus, one affecting ponderosa and Jeffrey pines in northern California and the other attacking singleleaf pinyon in southern California. The ponderosa form shows less vigor as a pathogen than the pinyon form, and on media in culture soon loses its capacity for conidial sporulation, whereas the pinyon form, both from California and Colorado, retains its fruiting capacity in culture over long periods.

From limited evidence, the fungus does not appear to retain its viability for long after the affected host or host part dies. However, in one case the organism was isolated from the root of a pinyon pine that had been dead for 10 years in Mesa Verde National Park.

INFECTION TESTS

Five series of tests to attempt the field infection of naturally established pines were undertaken, two at Blacks Mountain Experimental Forest in California and three in Mesa Verde National Park in Colorado. In the first at Blacks Mountain, slivers of green pinyon sapwood from a block overgrown in culture by an isolate of the fungus from singleleaf pinyon pine were inserted aseptically under the bark of carefully bared roots of ponderosa or Jeffrey pines 0.9 to 3.0 inches in stem diameter, or were wrapped in contact with small roots. On the last tree, a Jeffrey pine, the remainder of a sapwood block was buried in contact with the taproot. In the second series, 1 x 1 x 3 cm blocks of unseasoned pine root wood partially overgrown in culture by isolates of the fungus from singleleaf pinyon and Jeffrey pines were buried in contact with roots of ponderosa and Jeffrey pines 1.5 to 7 feet high.

At Mesa Verde, blocks about 2 inches long of invaded pinyon pine wood from a naturally infected tree were placed in contact with roots of 25 2-year-old pinyon seedlings and 25 of somewhat larger and older seedlings. In another series 2 years later, pieces of diseased root were placed in contact with roots of 25 pinyon seedlings 4 years old. A third series of older pinyon seedlings up to 2.5 feet in height was exposed the same way.

Infected slivers, when introduced into roots in the initial Blacks Mountain test, gave positive results in 14 out of 22 cases, but the infections were limited and did not cause a typical invasion by the fungus of the entire root system. Only where blocks with the fungus were placed in contact with roots without wounding did typical infections result, with death of the attacked trees within 1 to 2 years. In all, three young Jeffrey pines in California and eight pinyon seedlings in Colorado succumbed in the tests, a rather low percentage but enough to suggest that contact transfer from diseased to normal roots is the usual means of spread of the disease in nature.

---

[6] Letter June 1, 1960, W. B. Kendrick, Mycologist, Plant Research Institute, Central Experimental Farm, Ottawa, Canada.

[7] Personal communication.

RATE OF SPREAD IN PINYON WOODLANDS

To observe the progress of the disease in nature, three plots were established in single-leaf pinyon woodlands near Baldwin Lake in California and one in pinyon in Mesa Verde National Park, Colorado.

At both places an attempt was made to find locations where the disease was progressing from a newly established center, but only one of this type was found near Baldwin Lake and one at Mesa Verde. Plots were established around these centers. In addition, two plots (Nos. 2 and 3) were laid out across the irregularly advancing margins of older infection centers in the Baldwin Lake area. At Baldwin Lake, the number of trees dying from root disease on the three plots was as follows:

Plot	Before 1944	1944	1945	1946	1947	1948	1949	1950	Total
1	2	4	2	8	6	5	7	2	36
2	4	4	9	15	15	2	8	2	59
3	4	4	5	3	4	3	3	1	27
Totals	10	12	16	26	25	10	18	5	122

On the Mesa Verde plot in Colorado, mortality from disease was:

1942-1944	1945	1946	1947	1948	1949	1950	1951	1952	Total
5	0	2	1	0	4	7	5	6	30

Some trees survived both on the California and the Colorado plots. The escapes totalled 29 trees on the California plots, of which 62% were under 6 feet in height. On the Colorado plot, six trees within the zone of advance of the disease had apparently escaped infection. Most of the escapes are small trees with limited root systems that do not come in close contact with diseased roots. These escapes provide the nucleus for a new stand on the areas over which the disease has passed. In Mesa Verde National Park all stages of residual and replacement stands of pinyon in old disease areas can be found.

In California and Colorado the rate of progress of the disease through pinyon woodlands was about 40 feet in 6 years. The rate in ponderosa and Jeffrey stands could not be determined because in most places where the disease was discovered the timber stand was subsequently opened up by logging and the fungus apparently died out. The effect may be associated with increased soil temperatures unfavorable for the fungus after the removal of shade.

Particularly in Mesa Verde National Park, but to a lesser degree at Baldwin Lake also, mortality in pinyon pine from the disease was not constant from year to year but tended to be concentrated in irregular peak periods. At Mesa Verde, mortality was high in 1933 and 1934 and again in 1942 to 1944; it showed a moderate rise in 1952. Allowing a time lapse for infection to result in mortality, these increases appear to be associated with seasons of well-distributed summer rains, heavy enough to maintain good soil moisture levels through the season. Whether the moisture stimulates infection directly or through reducing soil temperatures or by both influences working together has not been determined.

DISCUSSION

No attempts to control the disease have yet been made and nothing is known of the means of transport of the fungus to initiate new infection centers. Two characteristics of the fungus should facilitate control in established centers of infection: it is not active as a saprophyte, and it grows well only in the low temperature range.

Evidence, including some not adduced here, indicates that the disease is essentially the same as that reported in Pinus strobus in Montana by Leaphart[2], and is probably induced by a strain of the same fungus. So far as known to us, it represents a new type of pathogenic root disease in forest trees.

PACIFIC SOUTHWEST FOREST AND RANGE EXPERIMENT STATION,
FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE,
BERKELEY, CALIFORNIA

PAXILLUS ATROTOMENTOSUS CAUSES BROWN ROOT ROT IN DEAD JACK PINE IN PLANTATIONS IN WISCONSIN

Ross W. Davidson[1] and Robert F. Patton[2]

In the summer of 1959, 25-year-old dying jack pine (Pinus banksiana) in plantations near Wisconsin Rapids in central Wisconsin were examined in an attempt to determine what fungi were associated with decay deterioration. There was little evidence of extensive decay in trees of such stands before death or decline started. Subsequent examinations indicated that in some stands decline of the trees was initiated through damage to the lateral root system by larvae of an unidentified "root tip weevil." In other stands trees with different symptoms died from attacks in the crown of a bark beetle, apparently Ips pini (Say). After death of the trees decay deterioration was fairly rapid. Some of the dead trees decayed and broke over above ground line and others tipped over by breakage of large roots below ground line.

Although no intensive study was made of decay deterioration of the dead trees, those falling over from breakage below ground line contained brown rot in the lower butt and root collar section and especially in the larger roots. The decay was typical of that usually referred to as brown carbonizing, with roots breaking sharply across the grain in typical manner for this type of decay. Dead trees with this type of decay occurred in both types of deteriorating stands described above.

In August sporophores of an agaric in close association with the brown root rot were collected frequently. Specimens sent to Dr. Alexander H. Smith, University of Michigan, were identified as Paxillus atrotomentosus Fr., a fungus reported to fruit on and around logs and stumps of trees in coniferous woods (4).

Meredith (5, 6) grouped it with the type of saprophytic stump fungi, such as Hypholoma fasciculare (Huds.) Fr. and Tricholoma rutilans (Schaeff.) Fr., which commonly invade stump roots by mycelium present in the soil or litter, as contrasted with "stump-surface colonizers." It was most abundant on pine stumps 5 years old or more. Kääarick and Rennerfelt (3) occasionally found sporophores of P. atrotomentosus at the base of 5-year-old spruce stumps. They believed it occurred more often than the presence of fruiting bodies would indicate and stated it (along with other agarics mentioned) had not yet been described in culture. There is, however, no record of the type of decay or its close association with pine roots. In the Wisconsin collections the prominent rhizomorphic yellow strands of mycelium extending from the base of stipes of the basidiocarps to the larger roots were characteristic, leaving little doubt regarding its causal relationship with the brown decay.

PURE CULTURES

Numerous spore prints were obtained both on sterile glass slides and by direct deposit on several types of culture media. None of the spores germinated even though some were maintained on the medium for periods of 3 or 4 weeks. It was also difficult to obtain growth of P. atrotomentosus from sterile tissue planting, but in one case a tissue culture was obtained. Also, one culture was isolated from brown rot in a root. These two cultures are similar, and the isolate from the brown root rot is believed to be P. atrotomentosus.

Growth in culture is very slow with the mycelium developing in a plumose to feathery mound over the inoculum. Meredith (6) also reported the fungus to be relatively slow growing, with the optimum growth temperature between the range of 22.5° and 25°C.

The substratum on malt or Hagam agar medium was darkly stained, almost black with the stain diffusing out into the medium beyond the mycelium. Dark stain also developed when it was transferred to malt agar medium containing gallic acid or tannic acid in small amounts. The stain in this case is not believed to be the ordinary oxidase reaction characteristic of white rot fungi as reported by several investigators including Davidson, et al. (1) (Fig. 1).

Technical Description of Cultures[3]

[1]Forest Pathologist, Beltsville Forest Disease Laboratory, Forest Service, United States Department of Agriculture.

[2]Associate Professor, Department of Plant Pathology, University of Wisconsin.

[3]The methods used in studying the cultures of this fungus, arrangement of description and explanation of key pattern are similar to those previously published (1, 2). Mat descriptions were based on 7- and 14-day-old Petri dish cultures grown at room temperature of approximately 25°C on 2 1/2% Fleischmanns diamalt agar in diffused light. Colors in quotation marks are according to Ridgway (7)

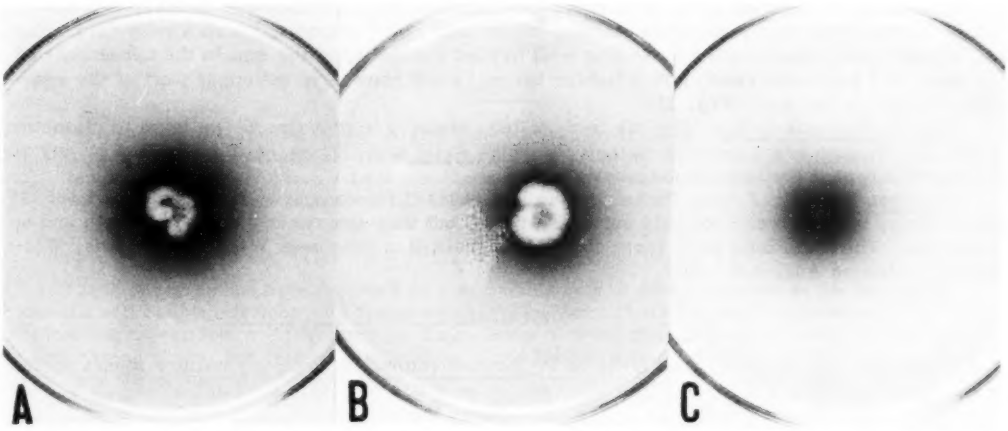


FIGURE 1. Photographs of cultures of Paxillus atrotomentosus Fr. on three media after 7 days' growth at 25° C. A -- Cultures on 2 1/2% diamalt agar medium. B -- Cultures on 2 1/2% diamalt agar medium with 0.5% gallic acid. C -- Cultures on 2 1/2% diamalt agar medium with 0.5% tannic acid.

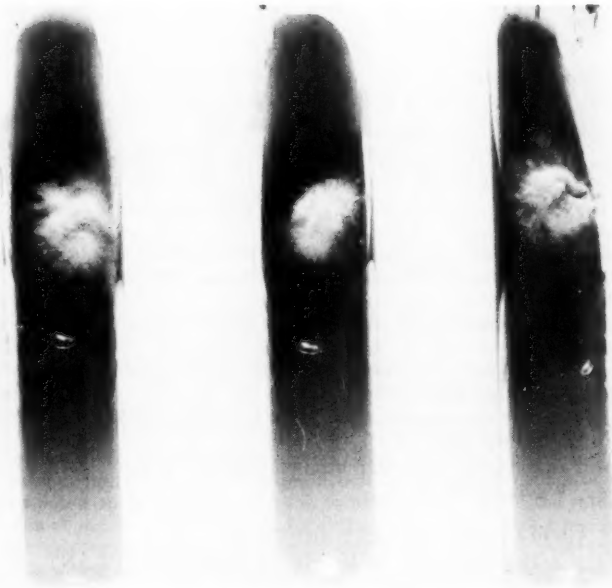


FIGURE 2. Test tube cultures of Paxillus atrotomentosus Fr. showing dark stain produced in the substratum

Key Pattern: C-O-V-1-10 C-O-S-1-10

Growth Characteristics: Growth slow, up to about 20 mm diameter in 14 days; mat mound-like over inoculum block, loose plumose to feathery, rather coarse in appearance, with narrow marginal zone appressed to agar medium; margin proper fimbriate; color of mat buff, "cream buff" to "warm buff," sometimes small brown "yellow-ocher" flecks are present over the mat from strands collapsed by guttation drops; guttation drops of clear slightly colored liquid numerous over the mycelial mat; substratum with pronounced dark brown to black stain under mat, "yellow ocher" to "buckthorn brown" and light red brown stain diffused out in agar medium 20 to 25 mm beyond margin of mycelium; trace of growth to good growth on gallic acid agar and no growth or only a trace on tannic acid agar medium, with pronounced darkening of substratum on both of these media (Fig. 1).

In test-tube cultures at 28 days the mat mounding over inoculum and covering only part of the surface of the slanted agar medium (about 22 mm in diameter), downy to feathery, thinning out to uneven somewhat appressed margin, color light buff "chamois," with a very dark "blackish brown" agar discoloration extending well beyond the limits of the mat in the upper part of the slant and gradually shading to a lighter brown "snuff brown" in the upper part of the agar cylinder (below the mat) (Fig. 2).

Hyphal Characteristics: Hyphae thin-walled, staining in phloxine B, uniform in diameter, 2 to 4.5μ, seldom branched with occasional cross walls and clamps, with no spores or any other outstanding hyphal features of diagnostic value.

Cultures Studied: Forest Pathology numbers 105331 (from sporophore tissue) and 105331 (from decay in root) were the only cultures studied but they are exactly alike in growth and appearance. The isolations were from collections from jack pine near Wisconsin Rapids, Wisconsin, made on August 7, 1959.

DISCUSSION

The dark stain formed by cultures of P. atrotomentosus in ordinary culture media makes the presence of such a dark reaction when grown on gallic or tannic acid unreliable as an indication of type of decay it produces. For this reason the key pattern as given indicates it is a brown rot species. It has been reported previously (1) that color reactions are not always reliable for indicating type of decay produced. As shown in the photographs, the discoloration is even more pronounced on malt agar medium than it is on the others shown.

Mycelial growth is sometimes good on medium containing gallic acid (Fig. 1); however, growth on this medium is usually less than on malt agar medium. The fungus makes no growth or only a trace on malt agar containing tannic acid.

Literature Cited

1. DAVIDSON, ROSS W., W. A. CAMPBELL, and DOROTHY J. BLAISDELL. 1938. Differentiation of wood-decaying fungi by their reaction on gallic or tannic acid medium. J. Agr. Research 57: 683-695.
2. DAVIDSON, ROSS W., W. A. CAMPBELL, and DOROTHY BLAISDELL VAUGHN. 1942. Fungi causing decay of living oaks in the eastern United States and their cultural identification. U. S. Dept. of Agr. Tech. Bull. 785: 65 pp.
3. KÄÄRICK, A., and E. RENNERFELT. 1957. Investigations on the fungal flora of spruce and pine stumps. Medd. Skogsforsknlnst., Stockholm 47(7): 88 pp.
4. KAUFFMAN, C. H. 1918. The Agaricaceae of Michigan (pages 287 and 288), 924 pp.
5. MEREDITH, D. S. 1959. The infection of pine stumps by Fomes annosus and other fungi. Ann. Bot. n.s. 23: 455-476.
6. MEREDITH, D. S. 1960. Further observations on fungi inhabiting pine stumps. Ann. Bot. n.s. 24: 63-78.
7. RIDGWAY, R. 1912. Color standards and color nomenclature. 43 pp. and 53 colored plates. Washington, D. C.

BELTSVILLE FOREST DISEASE LABORATORY, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF WISCONSIN, MADISON, WISCONSIN

STUDIES ON THE OCCURRENCE AND DISTRIBUTION OF THE OAK WILT FUNGUS IN OAK LEAVES[1]

H. C. Thompson[2]

Abstract

The oak wilt fungus, Ceratocystis fagacearum (Bretz) Hunt, was isolated from nearly half of the red oak leaves and from about a fourth of the white oak leaves collected from infected trees. Leaf inoculations with the oak wilt fungus produced oak wilt in more than half of the red oak trees that were inoculated.

INTRODUCTION

Little research has been reported on the possible role of infected leaves in the spread of the oak wilt fungus, Ceratocystis fagacearum (Bretz) Hunt. Using leaves from naturally infected oak trees, Henry (1) isolated the fungus from petioles of eight leaves, from midribs or larger veins of six, and from green portions of the blades of two. Henry and Riker (2) reported isolating the pathogen from 19 of 28 petioles, 6 of 20 midribs, 5 of 15 lateral veins, and the green portion of 2 of 20 blades from red and black oak leaves showing symptoms of infection. The fungus was also isolated by Henry and Riker from the petioles but not from other parts of 2 of 6 leaves showing no symptoms of infection from a wilting oak tree. Young (3) isolated the pathogen from petioles and midribs of red oak leaves showing symptoms of wilt.

There are few reports of research on leaf wounds as infection courts of the oak wilt fungus. Limited inoculation tests were made by Henry and Riker (2) who isolated the pathogen from 4 of 6 black oaks inoculated in the petioles of leaves. One of 13 trees inoculated in leaf blades developed oak wilt. In the greenhouse 1 of 9 red oak seedlings inoculated in the midrib developed the disease.

METHODS

To determine the distribution of the oak wilt fungus in oak leaves, the author collected leaves during the months of July, August, and October 1960, from both artificially inoculated and naturally infected red oaks, Quercus rubra, and white oaks, Q. alba. These trees were growing in nursery rows and were approximately 3 inches in diameter at breast height. Leaves showing no visible symptoms, leaves with partial bronzing, and leaves with complete bronzing were collected. Isolations were attempted from both the veins and from the interveinal tissue. Sections 1 cm long were cut with scissors from the petioles, from the midribs near the middle of the leaf blade, and from the largest lateral veins halfway between the midrib and the leaf margin. Squares of interveinal tissue approximately 5 x 5 mm were cut from near the central part of the leaf blade midway between the midrib and the leaf margin. The excised petioles, midribs, lateral veins, and interveinal tissues were placed in cheesecloth, washed in running tapwater for 3 minutes, and then surface sterilized for 1 minute in commercial Clorox diluted 50% with water. Each type of leaf material was treated separately. Small portions from the ends of each vein section were sliced off aseptically and each vein section was placed on potato-dextrose agar medium. The 5-mm square sections of interveinal tissue were drained and plated directly on agar medium.

In August 1960, 11 red oaks showing no symptoms of oak wilt were selected for leaf inoculation studies. These trees were growing approximately 5 feet apart in the nursery row and were approximately 2 to 3 inches d.b.h. On the undersides of the leaves of these trees wounds were made on the midribs, on the lateral veins, and in the interveinal tissue. Wounds on the leaves of six trees were made by cutting diagonally with a razor blade; wounds on the leaves of five trees were made by crushing small areas of tissue with a pestle. Two of the trees with leaves wounded by cutting, and two of the trees with leaves wounded by crushing received one wound on each of 12 leaves. Two of the trees with leaves wounded by cutting and two of the trees with leaves wounded by crushing received one wound on each of 24 leaves. The remaining two trees with leaves wounded by cutting and the one remaining tree with leaves wounded by crushing received one wound on each of 36 leaves. A third of the leaves were wounded on the midribs, a third on lateral veins, and a third in interveinal tissue. One drop of a conidial suspension made from a culture of the oak wilt fungus was placed in each wound immediately after wounding.

RESULTS

The oak wilt fungus was isolated more frequently from red oak leaf parts than from white oak leaf parts (Table 1). In all cases the pathogen was isolated from petioles more frequently

[1]The material included in this article is based upon a thesis submitted in partial fulfillment of the requirements for the M.S. degree at the University of Illinois.

[2]Research Assistant, Illinois Natural History Survey, Urbana, Illinois.

Table 1. Frequency of isolating the oak wilt fungus from leaves collected from wilting oaks.

Species of oak	Leaf symptoms	Part of leaf used	No. tested	% yielding fungus
	None	petiole	93	11.8
		midrib	51	5.9
		lateral vein	12	0
		interveinal tissue	15	0
Q. alba	Partial bronzing	petiole	93	17.2
		midrib	51	11.8
		lateral vein	12	0
		interveinal tissue	15	0
	Complete bronzing	petiole	27	22.2
		midrib	27	0
		lateral vein	12	0
		interveinal tissue	15	0
	None	petiole	573	41.4
		midrib	519	26.0
		lateral vein	79	2.5
		interveinal tissue	68	0
Q. rubra	Partial bronzing	petiole	114	56.1
		midrib	114	21.1
		lateral vein	18	0
		interveinal tissue	78	0
	Complete bronzing	petiole	68	32.4
		midrib	68	0
		lateral vein	18	0
		interveinal tissue	67	0

than from any other part of the leaf, midribs were second, followed by lateral veins. In no instance was the fungus isolated from interveinal tissue.

Leaves showing partial bronzing yielded the oak wilt fungus somewhat more frequently than leaves with no symptoms. Leaves with complete bronzing yielded the fungus least frequently. The fungus was isolated only from the petioles of leaves showing complete bronzing. It was isolated only from lateral veins of red oak leaves showing no symptoms of wilt.

Seven of the 11 red oak trees inoculated with a conidial suspension in the midribs, lateral veins, and interveinal tissue of leaves developed oak wilt. The fungus was isolated from the petiole of one leaf on a branch approximately a foot from the nearest inoculated leaf 2 weeks before symptoms appeared on the tree. The first symptoms developed on the inoculated leaves within 4 weeks. These symptoms were followed by symptoms on leaves in the upper crown of the trees. By the last week in September, 11 weeks after the trees were inoculated, symptoms were general throughout the trees from which the fungus was isolated. None of the four trees used for controls developed oak wilt.

DISCUSSION

The oak wilt fungus is present in oak leaves possibly more frequently than was formerly believed. From red oak and white oak leaves collected from infected oak trees the fungus was isolated from 37.2% of the leaves showing no wilt symptoms, from 38.6% of the leaves showing partial bronzing, and from 29.5% of the leaves showing complete bronzing. The fungus can be present in some leaves before any visible symptoms are evident. The possible importance of leaves serving as a reservoir for inoculum in the spread of the oak wilt fungus seems evident. It seems especially significant that infected white oaks may possibly serve as sources of inoculum since they may have infected foliage for a number of years before they die.

Wounds on leaves can serve as infection courts for the oak wilt fungus. To determine the importance of leaf wounds the role of leaf-feeding insects should be investigated further.

Literature Cited

1. HENRY, B. W. 1944. Chalara quercina n. sp., the cause of oak wilt. Phytopathology 34: 631-635.
2. HENRY, B. W., and A. J. RIKER. 1947. Wound infection of oak trees with Chalara quercina and its distribution within the host. Phytopathology 37: 735-743.
3. YOUNG, ROY A. 1949. Studies on oak wilt, caused by Chalara quercina. Phytopathology 39: 425-441.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF ILLINOIS, URBANA, ILLINOIS

THREE UNCOMMON WATERMELON FRUIT ROTS IN FLORIDA[1]

N. C. Schenck[2]

Abstract

The symptoms on watermelon fruits infected by Cylindrocladium scoparium (reported for the first time), Macrophomina phaseoli and Pellicularia rolfsii are described. All species, except M. phaseoli, infected nonwounded surfaces of watermelon fruits. C. scoparium caused damping-off of watermelon seedlings, infected nonwounded watermelon cotyledons, and within 24 hours penetrated stomata of watermelon rind. No damping-off or cotyledon infection resulted after inoculation with M. phaseoli or P. rolfsii.

Above normal rainfall in north Florida during watermelon harvest in June 1958 was especially conducive to fruit rot development and resulted in the rejection of more than the usual numbers of watermelons at loading stations. Most of the diseased fruits were infected with Mycosphaerella citrullina (C.O. Sm.) Gross., but several rotted fruits had lesions discernibly different from those caused by M. citrullina and Colletotrichum lagenarium (Pass.) Ell. & Halst., the predominant fruit rotting fungi in Florida.

The fungi isolated from these fruits were Cylindrocladium scoparium Morgan, Macrophomina phaseoli (Sclerotium bataticola) (Maubl.) Ashby, and Pellicularia rolfsii (Sclerotium rolfsii) (Curzi) E. West. C. scoparium has been reported infecting several hosts (1, 2, 3), but has never been reported from watermelon. M. phaseoli was reported on watermelon in Texas (5), but not described on the fruit. P. rolfsii was recently reported on watermelon fruits from Texas and Mexico (4).

This report describes the symptoms of infection on watermelon by these fungi and reports the occurrence of C. scoparium on watermelon for the first time.

MATERIALS AND METHODS

Diseased watermelon fruits were collected in several fields near Monticello, Florida and removed to the laboratory for diagnosis. After being rinsed with 70% ethyl alcohol, small pieces of diseased tissue were removed aseptically from the advancing edge of lesions and placed in moist chambers for observation, or onto acidified potato-dextrose agar and water agar in Petri plates. Pure culture isolations from such platings were used to make inoculations.

Watermelon fruits were selected from the field for freedom from injuries and naturally-occurring rots. Areas of melons intended for nonwounding inoculation were carefully examined to avoid tears or punctures in the cuticle. Melons were washed in tap water and rinsed in 70% ethyl alcohol prior to inoculation. Inoculations were made, using aqueous suspensions of mycelia and/or spores or small pieces of agar culture of the fungi, on both wounded and nonwounded surfaces of such melons. Wounds were made by pricking the melon surface to a depth of 1 mm with a sterile needle. Inoculated areas were then covered with several layers of moist cheesecloth and covered with clear polyethylene film affixed to the melon with cellophane tape. Inoculated melons were held in the laboratory (approximately 72° F) for observations of fruit rot development.

Twenty ml of an aqueous suspension of mycelia and/or spores of each organism were poured over sterile soil in each of three pots containing 10 watermelon seeds each. Seedling emergence and damping-off were observed at regular intervals. During this test greenhouse temperatures averaged 68° F.

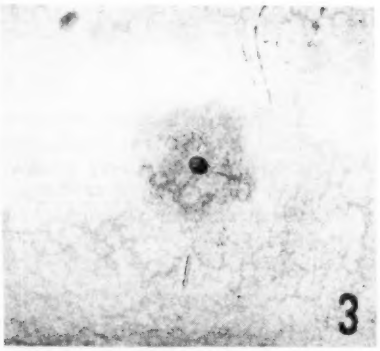
Small pieces of agar culture and mycelial or spore suspensions of each organism were placed on both wounded and nonwounded cotyledons of four watermelon seedlings growing in a 4-inch clay pot. The pots of inoculated seedlings were then covered with 1-quart clear polyethylene bags and examined after 48 hours for lesion development.

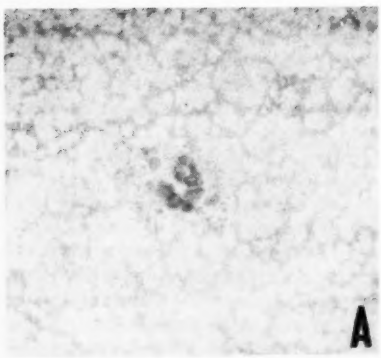
Several drops of a spore suspension of C. scoparium, the only fungus producing spores abundantly, were placed on pieces of melon rind 1 inch thick. After 12, 16, and 24 hours' incubation in a moist chamber, slices of epidermis (75μ thick) were removed with a sharp scalpel,

[1]Florida Agricultural Experiment Station Journal Series, No. 1308.

[2]Assistant Plant Pathologist, Watermelon and Grape Investigations Laboratory, Florida Agricultural Experiment Stations, Leesburg, Florida.

FIGURE 1. Watermelon seedlings (variety Charleston Gray) infected at the soil level and on the cotyledons with Cylindrocladium scoparium.





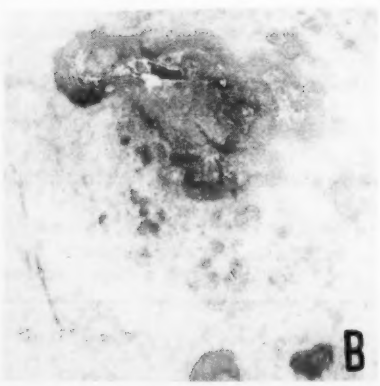


FIGURE 2. (left) Watermelon fruit (variety Charleston Gray) infected with Cylindrocladium scoparium. A -- Nonwounded surface 4 days after inoculation. (Note secondary infections surrounding initially infected area.) B -- Advanced lesions 14 days after inoculation.

FIGURE 3. Watermelon fruit (variety Charleston Gray) 4 days after inoculation (following wounding) with Macrophomina phaseoli.

FIGURE 4. Lesion 7 days after inoculation with Pellicularia rolfsii on a nonwounded surface of watermelon fruit (variety Charleston Gray).

placed for 1 minute in Wayson's Stain, rinsed with deionized water and examined under the microscope for germ tube development and penetration.

RESULTS

All species produced lesions on watermelon fruits and all but M. phaseoli penetrated without prior wounding (Table 1). After 24 hours C. scoparium abundantly penetrated stomata and wounds. Germ tube growth was extensive and frequently hyphae from one germ tube entered more than one stoma.

Table 1. Lesion development on Charleston Gray melons 7 days after inoculation with three fruit-rotting fungi.

	Diameter of lesion (mm)			
	Agar culture		Spore or mycelial suspension	
Organism	wound	no wound	wound	no wound
Cylindrocladium scoparium	22	18	12	8
Pellicularia rolfsii	11	0	14	9
Macrophomina phaseoli	70	0	55	0

Damping-off occurred in 12 of 17 emerged seedlings after inoculation with C. scoparium (Fig. 1). No damping-off occurred after inoculation with the other two species.

All cotyledons inoculated with C. scoparium were infected regardless of wounding. Lesions were small and tan to reddish in color; in older lesions, bristle-like mycelium and sporophores were evident. P. rolfsii caused a small water-soaked lesion on only one wounded cotyledon.

DESCRIPTION OF FRUIT ROTS

C. scoparium caused a slowly spreading dry rot on watermelon fruits. Lesions first appeared as small (1 to 2 mm in diameter), circular, raised, water-soaked areas, the centers of which became sunken and tan in color with further development. Large lesions usually resulted from the coalescence of several smaller lesions. Older lesions were sunken, cracked, irregular to rectangular in shape, brown to reddish tan in color but with a dark green, raised, water-soaked border (1 to 2 mm wide) and usually with prominent bristle-like mycelia and sporophores (Fig. 2).

Lesions produced by M. phaseoli were round to irregular in shape and dark green to tan in color, with light green water-soaked convolute edges (2 to 4 mm wide). Lesions developed rapidly, up to 32 mm in diameter in 3 days and up to 100 mm in 7 days. The rot was not sharply delimited in color or appearance from healthy areas until the later stages, when sclerotial development blackened the center of the lesions and cracks developed throughout the lesioned area (Fig. 3).

P. rolfsii produced a soft rot, light tan to gray in color, with darker water-soaked edges (about 2 mm wide). Lesions were slightly sunken, 5 to 10 mm in diameter after 3 days and up to 20 mm after 7 days. Cracks, emanating from the center, generally with white mycelium, yellow ooze, and sclerotia, developed readily in lesions over 10 mm in diameter (Fig. 4).

Literature Cited

1. ANDERSON, P. J. 1918. Rose canker and its control. Massachusetts Agr. Exp. Sta. Bull. 183. pp 11-46.
2. COX, R. S. 1954. Cylindrocladium scoparium on conifer seedlings. Delaware Agr. Exp. Sta. Tech. Bull. 301. 40 pp.
3. KATZNELSON, H. and L. T. RICHARDSON. 1948. Rhizosphere studies and associated microbiological phenomena in relation to strawberry root-rot. Sci. Agr. 28: 293-308.
4. RAMSEY, G. B., M. A. SMITH, L. BERAHA, and W. R. WRIGHT. 1959. Pellicularia rolfsii on Mexican and Texas watermelon. Plant Disease Reptr. 43: 1031
5. YOUNG, P. A. 1944. Epidemic of charcoal rot of corn and other crops in east Texas. Plant Disease Reptr. 28: 898-899.

WATERMELON AND GRAPE INVESTIGATIONS LABORATORY,
FLORIDA AGRICULTURAL EXPERIMENT STATIONS, LEESBURG, FLORIDA

OBSERVATIONS ON NEW OR UNUSUAL DISEASES OF HIGHBUSH BLUEBERRY

L. C. Raniere[1]

Unusually cool, wet weather during the 1960 growing season and very low temperatures the following winter throughout most of northeastern United States are thought to have contributed to the occurrence of previously unobserved blueberry diseases.

ROOT AND CROWN ROT

Rooted blueberry cuttings with leaf yellowing and early defoliation from the propagation beds of two nurseries in New Jersey were examined in August and September 1960. Affected plants showed some root necrosis and extensive vascular discoloration in the crowns and main stems. Large numbers of Coville plants were killed, but Weymouth plants in adjacent nursery rows were unaffected.

Many tissue transfers to potato-dextrose agar revealed one conspicuous fungus. The rapid growth of characteristic white concentric mycelial colonies suggests a species of Phytophthora but all attempts to obtain asexual or sexual sporulation have failed thus far. A culture was sent to C. J. Hickman of the University of Western Ontario, who has encountered similar difficulties and until this time has been unable to identify this Phycomycete.

A circular area of dwarfed and dead plants in the corner of a young Earliblue planting was observed in July 1961. These plants were affected in varying degrees ranging from slight reduction in new growth to winter killing of previously weakened plants. Isolations from discolored crowns again revealed the presence of the Phytophthora-like fungus.

Further attempts will be made to identify this organism, determine its pathogenicity, and study varietal susceptibility.

SUMMER CANE BLIGHT

The sudden occurrence of extensive cane blighting was observed in many blueberry plantings from late June 1961 through the summer. In some cases apparently healthy canes died suddenly in 2 or 3 days. The dead leaves remained attached, and in most cases the vascular system was discolored the whole length of the affected canes.

Several attempts were made to isolate causal organisms from the margins of discoloration in the wood. Specimens from various areas in New Jersey, Michigan, Illinois and Ohio commonly yielded a fungus closely resembling Botryosphaeria ribis, which produced perithecia abundantly on recently killed wood. Close examination indicated that it differs from the description of the unidentified species of Botryosphaeria that causes a similar stem blight in North Carolina.

Occasionally Phomopsis vaccinii and an undescribed species of Gloeosporium were consistently isolated from blighted canes. P. vaccinii is frequently associated with dieback, but Gloeosporium species have been observed on only mature blueberry fruit and in small lesions or cankers on 1-year-old twigs. This organism is unusual on mature canes.

The severe winter temperatures had a direct effect by killing fruit buds, reducing fruit set and size, and killing branches of blueberry plants in 1961. Low temperature damage may have also predisposed many plants to invasion by various weak pathogens normally unable to cause any measurable injury to vigorous plants.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, NEW BRUNSWICK, NEW JERSEY

---

[1]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, New Jersey Agricultural Experiment Station, New Brunswick.

FUSARIUM SOLANI F. PISI, A CAUSE OF PEA ROOT ROT IN NEW HAMPSHIRE[1, 2]

Graydon C. Kingsland and Avery E. Rich[3]

Root rots are a limiting factor in the production of garden peas, Pisum sativum, in some areas of New Hampshire. Heavy losses frequently occur, and in some fields root rotting fungi cause complete crop failure.

Preliminary field observations suggested that the principal disease might be wilt, caused by Fusarium oxysporum f. pisi (Linford) Snyd. & Hans. race 1, or near wilt, caused by F. oxysporum f. pisi (Linford) Snyd. & Hans. race 2 (3, 5). However, cultivars which are resistant to wilt and near wilt were found to be susceptible to attack by the pathogen.

More detailed field and laboratory examinations indicated that brown to black cortical lesions developed on the roots and lower stem portions of affected plants. These symptoms occurred early in plant ontogeny and increased in severity with plant maturity. Girdling of the cortex of the main root and lower stem was ultimately complete. Leaf wilt and chlorosis, followed by death of the plants, due to interference with water conduction and nutrient translocation (6), developed about a week prior to fruit maturity in most varieties. This syndrome indicated a root rot rather than wilt. Fungi that frequently cause root rots of peas beyond the seedling stage include Aphanomyces euteiches Drechs., Pellicularia filamentosa (Pat.) Rogers, and F. solani (Mart.) Appel & Wr. f. pisi (F. R. Jones) Snyd. & Hans. These three fungi attack the cortical tissues of the roots and basal portions of infected plants (1, 2, 6). The injury they cause is so similar that the causal agent cannot be identified accurately by macroscopic observation of the host plants. Therefore, Koch's postulates were followed to determine the identity of the microorganism that was pathogenic on garden peas in these tests.

Roots and stems of peas showing various stages of disease development were cut into pieces about 0.75 cm in length. The sections were washed for 5 hours beneath a cold water spray to remove surface contaminants, then they were transferred to sterile water agar in Petri plates with flamed forceps. The fungi that grew from the plant tissues were later transferred to corn meal agar (4). The only fungus obtained consistently was a Fusarium, identified as F. solani by Dr. H. N. Hansen of the University of California at Berkeley. Relegation of this fungus to its subspecific taxonomic status required proof of pathogenicity.

The F. solani from infected peas was cultured in quart jars of sterile sand and corn meal medium (4). Equal amounts of the inoculum thus obtained were mixed into the upper half of five 10-quart pails of sandy loam soil which had been autoclaved in trays at 20 psi for 45 minutes. The surface of the soil in these pails was divided into four sections, and six seeds each of Progress No. 9, Little Marvel, Thomas Laxton, and New Era peas were planted in separate sections of each of the pails. This arrangement provided five replicates of each cultivar. The pails were placed on a greenhouse bench and maintained at a day temperature of about 25°C and a night temperature of about 20°. Within 23 days typical root rot symptoms (1) developed on the first three cultivars. New Era exhibited a degree of resistance to and considerable tolerance of infection.

A Fusarium was readily isolated from diseased portions of all varieties. This fungus appeared to be identical with original isolates which had been identified as F. solani. Pathogenicity of F. solani on garden peas relegates the fungus to F. solani f. pisi.

Literature Cited

1. BUTLER, E. J., and S. G. JONES. 1949. Foot rot of peas. In Plant Pathology, MacMillan and Co., London. pp. 620-623.
2. JONES, FRED REUEL, and CHARLES DRECHSLER. 1925. Root rot of peas in the United States caused by Aphanomyces euteiches (n.sp.). J. Agr. Research 30: 293-325.
3. LINFORD, M. B. 1928. A Fusarium wilt of peas in Wisconsin. Wisconsin Agr. Exp. Sta. Research Bull. 85.
4. RIKER, A. J., and R. S. RIKER. 1936. Introduction to research on plant diseases. John Swift and Co., St. Louis, Missouri.
5. SNYDER, W. C., and J. C. WALKER. 1935. Fusarium near wilt of peas. Zbl. Bakt. Abt. 2, 41: 355-378. (Abst. Rev. Appl. Mycol. 14: 486. 1935).
6. WALKER, J. C., and W. C. SNYDER. 1933. Pea wilt and root rots. Wisconsin Agr. Exp. Sta. Bull. 424.

NEW HAMPSHIRE AGRICULTURAL EXPERIMENT STATION, DURHAM

[1]Published with the approval of the Director of the New Hampshire Agricultural Experiment Station as Scientific Contribution No. 277.
[2]Based on a portion of a thesis submitted by the senior author to the Graduate School, University of New Hampshire, in partial fulfillment of the requirements for the degree of Master of Science.
[3]Assistant Plant Pathologist, Department of Botany and Bacteriology, Clemson College, Clemson, South Carolina (Formerly Graduate Research Assistant, New Hampshire Agricultural Experiment Station); and Plant Pathologist, New Hampshire Agricultural Experiment Station, respectively.

RESISTANCE IN TRITICUM VULGARE TO INFECTION BY ERYSIPHE GRAMINIS F. SP. TRITICI AS INFLUENCED BY THE STAGE OF DEVELOPMENT OF THE HOST PLANT

L. W. Briggle and A. L. Scharen[1]

Summary

Several varieties and lines of wheat, Triticum vulgare (Triticum aestivum), are susceptible as seedlings to Erysiphe graminis DC. f. sp. tritici Em. Marchal but are resistant at later stages of development. This type of resistance has been considered adequate in a commercial variety. Five such varieties and lines of wheat were inoculated at various stages of growth with two cultures of E. graminis tritici. Their reactions were related to the stage of development of the plants. All were susceptible in the seedling stage and resistant at later stages of development. All studied were resistant to culture 6 when they had attained the jointing stage of development. Redcoat was resistant to culture 116-2 in the rosette stage, but Coker 55-3 was not resistant until the boot stage. The reaction of tillers depends on their stages of growth rather than on the stage of the older culms.

INTRODUCTION

Some varieties and breeding lines of wheat, Triticum vulgare (Triticum aestivum), are highly resistant at all stages of development to infection by specific cultures of Erysiphe graminis DC. f. sp. tritici Em. Marchal. Others are susceptible at all stages. A few wheats susceptible in the seedling stage are resistant at later stages of development. This adult-plant resistance has been adequate for a commercially grown wheat.

Little, however, is known concerning the time, in terms of development of the wheat plant, at which adult-plant resistance to the powdery mildew organism is expressed. Available information has been based on general observations. However, the literature does contain several papers dealing with adult-plant resistance to other mildews and non-related pathogens. Graf-Marin (1) found adult-plant resistance in barley, Hordeum vulgare, that was inoculated with E. graminis DC. f. sp. hordei Em. Marchal when plants were in the late jointing to flowering stages. On the other hand, Schnathorst (5) states that powdery mildew of lettuce is primarily a disease of mature plants, the young plants being resistant. Newton and Johnson (4) in a study of adult-plant resistance in wheat to races of P. recondita Rob. ex Desm. showed that several varieties susceptible in the seedling stage were resistant in the heading stage. In the adult plant the resistance was greatest in the uppermost leaves but diminished progressively on the lower leaves. Johnston and Melchers (2) had earlier shown the same results with different varieties of wheat.

The study reported here was initiated to determine the stage of development at which adult-plant resistance to cultures of E. graminis tritici was first expressed. Differences in varietal response were a matter of primary interest.

MATERIALS AND METHODS

Two varieties of soft red winter wheat, Redcoat (C. I. 13170)[2] and Coker 55-3 (C. I. 13250), which are susceptible in the seedling stage and highly resistant in the adult-plant stage to cultures 6 and 116-2 of E. graminis tritici, were planted outdoors in soil beds in the fall of 1959 at Beltsville, Maryland. Early in January, four plants of each variety were dug up and replanted in pots in the greenhouse. Two plants of each variety were placed in a constant-temperature greenhouse at 20° ± 2° C, and the other two were placed in an alternating-temperature greenhouse held at 13° ± 2° at night and 16° ± 2° during the day. After 6 weeks, the temperature in the latter greenhouse was gradually raised to 16° ± 2° at night and 20° ±2° during the day. Greenhouses were maintained at these temperatures until all plants were removed to growth chambers for inoculation with conidia of E. graminis tritici.

At approximately 1-week intervals from January through April, additional plants of the two varieties were transplanted to the greenhouse. This procedure resulted in an array of stages of development in the plants ranging from seedlings through ripening (Fig. 1).

---

[1]Research Agronomist and Pathologist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2]C. I. refers to Cereal Investigations number assigned by the Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

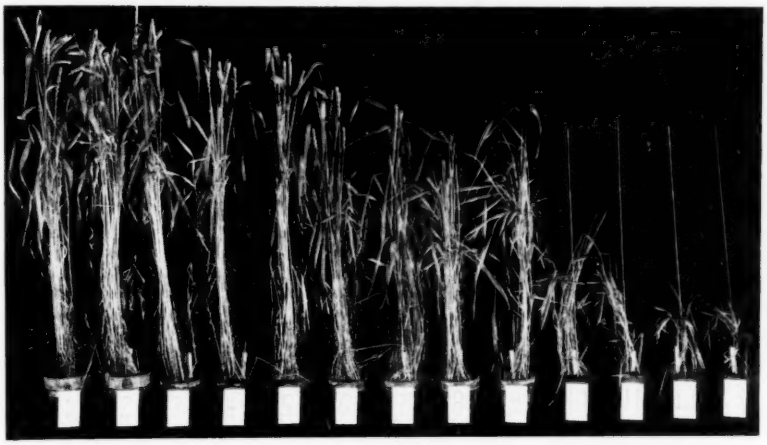


FIGURE 1. Stages of development of host plants inoculated with cultures of Erysiphe graminis tritici.

In April half the plants were moved to a growth chamber maintained at 20° ± 1° C. One complete array of each variety was from the constant-temperature greenhouse; a second array of each was from the alternating-temperature greenhouse. These plants were immediately inoculated with conidia of culture 116-2 of E. graminis tritici. Ten days after inoculation, readings were made on the reaction of plants in all stages of growth to the organism. The same type of test was conducted using the remaining array of wheat plants and culture 6 of powdery mildew.

The experiment was repeated in 1961 without change in procedure. In addition to Redcoat and Coker 55-3, the following varieties and lines of wheat were tested: Ramona 50 (C. I. 12390), Michigan Amber (C. I. 11170), and an Iowa selection (C. I. 13279). Only culture 6 of E. graminis tritici was used in 1961.

Seedlings were grown in the greenhouse and added to the array of developmental stages of all varieties both years. Stages of plant development given in Tables 1, 2 and 3 were as of the time of inoculation. Infection types used were those described by Mains and Dietz (3).

RESULTS

Representative reactions of Redcoat and Coker 55-3 plants at different stages of development are given in Tables 1 and 2. Resistance of Redcoat of cultures 116-2 and 6 of the powdery mildew fungus was evident at an early stage of growth. While seedlings were susceptible to both cultures, resistance was evident from the rosette stage to maturity in the 1960 tests. In the 1961 tests, resistance was not expressed until late in the jointing stage. Thus, Redcoat showed effective resistance to two cultures of E. graminis tritici under conditions of growth from the jointing stage to maturity. Culture 6 was slightly more virulent on Redcoat than was culture 116-2.

Resistance of Coker 55-3 to the two cultures of the organism became evident at a later stage of growth than that of Redcoat. When inoculated with conidia of culture 116-2, plants grown in the constant-temperature greenhouse were susceptible through the early heading stage and those grown in the alternating-temperature greenhouse did not express resistance until the boot stage. Resistance to culture 6 was evident in the boot stage. Plants were completely resistant in the post-flowering stage. A summary of results obtained for all varieties and lines is given in Table 3.

In all cases, tillers reacted according to their stage of development at the time of inoculation rather than in the same manner as the more mature main culms of a plant. For example, the principal culms of some plants in the post-flowering stage of development were free of mildew while tillers in the early jointing stage were susceptible.

DISCUSSION AND CONCLUSIONS

The wheat varieties and lines studied in this series of experiments were susceptible in early stages of development to two cultures of E. graminis tritici but were resistant after the jointing to boot stage had been attained. Apparent freedom from infection was evident if inoculations were made after late jointing, boot, or early heading stages had been reached, depending on the

Table 1. Reaction of Redcoat wheat at different stages of development under two temperature regimes to two cultures of E. graminis tritici.

Temperature regime, culture, year, and stage of plant development	Reaction	Temperature regime, culture, year, and stage of plant development	Reaction
Constant:		Alternating:	
Culture 116-2 (1960):		Culture 116-2 (1960):	
3-4 leaves [a]	4	3-4 leaves	4
Rosette	0; (tillers = 1-2)	Rosette	0-1
Rosette	0; (tillers = 1)	Rosette	0-1
Very early jointing	0;	Rosette	0-1
Early jointing	0;	Very early jointing	0-1
Jointing	0;	Early jointing	0-1
Early heading	0;	Jointing	0;-1
Headed	0 (tillers = 1)	Jointing	0-1
Headed	0 (tillers = 1)	Jointing	0-1
Milk	0 (tillers = 1)	Late jointing	0
Milk	0 (tillers = 1)	Late jointing	0
Dough	0 (tillers = 1)	Early boot	0
Dough	0 (tillers = 1)	Late boot	0
Hard dough	0 (tillers = 1)	Early heading	0
Culture 6 (1960):		Culture 6 (1960):	
3-4 leaves	4	3-4 leaves	4
Rosette	2	Rosette	1-2
Early jointing	1-2	Early jointing	1-2
Jointing	1	Jointing	1-2
Jointing	0-1	Jointing	1-2
Headed	0	Jointing	0-1
Milk	0	Late jointing	0-1
Dough	0	Early boot	0
Dough	0	Boot	0
Hard dough	0	Heading	0
Ripe	0	Headed	0
Ripe	0	Seed set	0
Ripe	0	Milk	0
Ripe	0	Dough	0
Culture 6 (1961):		Culture 6 (1961):	
3-4 leaves	4	3-4 leaves	4
---	-	Rosette	4
---	-	Early jointing	4
Early jointing	0;-1	Jointing	4
Jointing	0;-1	Late jointing	0;-2
Late jointing	0;-1	Boot	0;
Early heading	0;-1	Early heading	0;
Headed	0;-1	Headed	0; (Few glume pustules)
Seed set	0;	Seed set	0;
Milk	0;	Milk	0;
Soft dough	0;	Milk	0;
Dough	0 (tillers = 1)	Early dough	0
Ripe	0 (tillers = 1)	Dough	0

[a] Development is described in terms of plant growth for those plants not yet headed and in terms of seed maturity for plants after heading. Rosette = dormant stage before active elongation. Jointing = period of rapid elongation of internodes. Boot = stage in which upper leaf sheath becomes swollen, just prior to emergence of the spike. Early heading = tips of spikes visible. Heading = spikes emerged. Headed = anthers exserted. Seed set = first few days after pollination. Milk = seeds very soft and filled with milky fluid. Dough = endosperm developed beyond fluid consistency and is dough-like in composition. Ripe = head is no longer green and endosperm is semi-hard or hard.

Table 2. Reaction of Coker 55-3 wheat at different stages of development under two temperature regimes to two cultures of E. graminis tritici.

Temperature regime, culture, year, and stage of plant development	Reaction	Temperature regime, culture, year, and stage of plant development	Reaction
Constant:		Alternating:	
Culture 116-2 (1960):		Culture 116-2 (1960):	
3-4 leaves	4	3-4 leaves	4
Rosette	4	Rosette	4
Rosette	4	Rosette	4
Early jointing	4	Very early jointing	4
Early jointing	4	Very early jointing	4
Jointing	3-4	Early jointing	4
Late boot	3-4	Jointing	4
Early heading	3	Jointing	4
Headed	1 (tillers = 3-4)	Late jointing	4
Headed	0; (tillers = 3-4)	Boot	3-4
Seed set	0; (tillers = 3-4)	Late boot	0-1 (tillers = 3-4)
Milk	0;	Early heading	0; (tillers = 3-4)
Dough	0;	Early heading	0; (tillers = 3-4)
Dough	0;	Headed	0; (tillers = 3-4)
Culture 6 (1960):		Culture 6 (1960)	
3-4 leaves	4	3-4 leaves	4
Rosette	4	Rosette	4
Early jointing	4	Early jointing	4
Jointing	4	Early jointing	4
Late jointing	3	Jointing	4
Late boot	Tr-1 (tillers = 3)	Late jointing	4
Headed	Tr-1 (tillers = 3)	Early boot	4
Seed set	Tr-1 (tillers = 3)	Early boot	4
Milk	0 (tillers = 3)	Boot	0-Tr
Dough	0 (tillers = 1-2)	Headed	0-Tr
Dough	0 (tillers = 1-2)	Seed set	0 (tillers =3)
Late dough	0 (tillers = 1)	Milk	0 (tillers = 3)
Ripe	0 (tillers = 1)	Dough	0 (tillers = 3)
Ripe	0	Dough	0 (tillers = 3)
Culture 6 (1961):		Culture 6 (1961):	
3-4 leaves	4	3-4 leaves	4
---	-	Rosette	4
---	-	Early jointing	4
Early jointing	4	Jointing	4
Jointing	4	Jointing	4
Late jointing	4	Late jointing	0;
Boot	0;-2	Early boot	0;
Heading	0;-2	Early heading	0;
Headed	0;-2	Heading	0;
Seed set	0;-2	Headed	0
Milk	0; (tillers = 2)	Seed set	0
Dough	0; (tillers = 2)	Milk	0 (Pustules on glumes)
Dough	0; (tillers = 2)	Milk	0

variety. However, this same degree of resistance was not evident in plants grown under adverse temperatures. Four of the varieties and lines grown under constant temperature, a condition having a deleterious effect on plant vigor, were not resistant until later developmental stages when compared with the same varieties or lines grown under more optimal conditions. Freedom from mildew pustules was not evident until the post-flowering stage of development. Thus, the use of different growing conditions gave a gross evaluation of the effect of environment on resistance to E. graminis tritici. Plants grown in the alternating-temperature greenhouse can generally be compared with field grown plants in a normal season.

Table 3. Stage of plant development at which resistance to two cultures of E. graminis tritici is expressed in five varieties and lines of wheat grown in greenhouse under two temperature regimes.

Temperature regime, culture, and variety name	Stage giving indicated type of resistance	
	effective[a]	complete[b]
Constant:		
Culture 116-2:		
Redcoat	Rosette	Rosette
Coker 55-3	Heading	Heading
Culture 6:		
Redcoat	Early jointing	Headed
Coker 55-3	Late boot	Headed
Ramona 50	Early heading	Headed
Michigan Amber	Early jointing	Jointing
Iowa selection	Early jointing	Headed
Alternating:		
Culture 116-2		
Redcoat	Rosette	Jointing
Coker 55-3	Boot	Very early heading
Culture 6:		
Redcoat	Late jointing	Boot
Coker 55-3	Late jointing	Early boot
Ramona 50	Early jointing	Early boot
Michigan Amber	Jointing	Late jointing
Iowa selection	Early jointing	Early boot

[a]"Effective resistance" was considered to be expressed when a limited number of type 1 and 2 pustules were present. Any plant on which type 3 and 4 pustules were found was considered susceptible.
[b]"Complete resistance" indicates the absence of any pustules on the plant; a type 0 reaction.

The observation that tillers in an early stage of development may be susceptible to infection when the main culms of the same plant are highly resistant indicates that the type of resistance studied here is wholly dependent upon the stage of development of the particular tissue being parasitized. Young leaves on a single culm may not be as resistant as older leaves on the same culm. Graf-Marin (1) has shown that mature plant resistance to E. graminis hordei in barley is due to a thickening and toughening of the epidermal and cuticular tissues. Conversely, Schnathorst (5) has shown that the thickness of the epidermis and cuticle is not responsible for adult-plant resistance of lettuce, Lactuca sativa, to Erysiphe cichoracearum DC. ex Mérat. Further studies to elucidate the nature of the resistance discussed in this paper would be of interest.

Literature Cited

1. GRAF-MARIN, A. 1934. Studies on powdery mildew of cereals. Cornell University Agr. Exp. Sta. Mem. 157. 48 pp.
2. JOHNSTON, C. O., and L. E. MELCHERS. 1929. Greenhouse studies on the relation of age of wheat plants to infection by Puccinia triticina. J. Agr. Research 38: 147-157.
3. MAINS, E. B., and S. M. DIETZ. 1930. Physiologic forms of barley mildew, Erysiphe graminis hordei Marchal. Phytopathology 20: 229-239.
4. NEWTON, MARGARET, and T. JOHNSON. 1943. Adult plant resistance in wheat to physiologic races of Puccinia triticina Erikss. Can. J. Research C, 21: 10-17.
5. SCHNATHORST, W. C. 1959. Resistance in lettuce to powdery mildew related to osmotic value. Phytopathology 49: 562-571.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

DISTRIBUTION OF SOME CUCURBIT VIRUSES IN SOME SOUTHWEST MELON-PRODUCING AREAS

R. E. Webb[1]

Summary

A limited survey of cucurbit plantings in the Rio Grande Valley and Wintergarden areas of Texas, Mesa, Arizona, and El Centro, California indicated widespread virus infection in all areas sampled. Tobacco ringspot virus (TRSV) was the most prevalent virus present in cucurbit plantings in both areas in southern Texas and often occurred in mixtures with watermelon mosaic virus (WMV). Cucumber mosaic virus (CMV) and squash mosaic virus (SMV) were identified in only 9 and 6 specimens, respectively, of 155 assayed. The most prevalent virus in the Mesa, Arizona, and El Centro, California plantings was WMV. CMV was isolated from 21 of 40 specimens from the Mesa planting but was detected in only 8 of 45 specimens assayed from El Centro. SMV was identified in 7 of 40 specimens from Mesa and in one of 45 specimens from El Centro. TRSV, the most widespread cucurbit virus in southern Texas, was not detected in cucurbit specimens from El Centro or cantaloup specimens from Mesa.

Several distinct viruses of the mosaic group are known to infect cucurbits under field conditions (2, 3). Among the more important of these in the United States are cucumber mosaic virus (CMV), squash mosaic virus (SMV), tobacco ringspot virus (TRSV), and watermelon mosaic virus (WMV) (1, 4, 5, 7, 10). Two or more of these viruses occur in most cucurbit-production areas and their relative prevalence varies in these areas as well as between localities (1, 4, 7, 10). In many instances, two or more viruses may be present in a field or even in the same plant (4, 7, 11).

In recent years melon, squash, and cucumber growers in the Southwest have experienced a sharp decline in production. Lack of fruit production or production of deformed and off-color fruit of watermelons, squash, and cucumbers due to widespread virus infection have become of great concern to growers. Also, a sharp decline in cantaloup and honeydew production has discouraged growers from planting these crops in the Rio Grande Valley of Texas, the Salt River Valley and Yuma Valley of Arizona, and the Imperial Valley of California. Melon plantings in these areas that do not produce profitable crops are usually affected with the so-called "crown blight." At the onset of the disease, discoloration and dead areas appear in the leaves at or near the crown of affected plants. The leaves gradually die forming an area of dead leaves more or less around the crown of the plant. Death of leaves outward along the runners may occur. In some instances, particularly near harvest time when temperatures are high, affected plants may suddenly wilt and die, particularly in Arizona and California.

Concurrent with the development of crown-leaf decline and death in melon plants, observations and surveys (4, 5, 7, 9) have indicated widespread virus infection. In Texas, death of crown leaves, moderate to severe retarded plant growth, and poor yields of midseason and late-planted melons are usually accompanied by virus infection. In Arizona and California virus infiltration into melon plantings usually coincides with the peak of aphid (primarily Myzus persicae Sulz.) migration. Crown-leaf decline and subsequent death of plants usually occur just before and during harvest.

A limited survey of melon plantings in Texas, Arizona, and California was conducted to determine the identity of viruses present, and their relative prevalence in the sampled areas.

MATERIALS AND METHODS

Virus-infected specimens of stems and leaves of honeydew and varieties of the cantaloup-type muskmelon, cucumber, squash and watermelon collected[2] for the most part at random in the field during the spring and fall of 1960 were received via airmal in small plastic bags. Each specimen was rinsed thoroughly in running tap water and blotted dry and a portion was sharp-

[1]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Maryland.

[2]Specimens from Mesa, Arizona were supplied by Robert Foster; from Crystal City, Texas by Bruce A. Perry; from the Rio Grande Valley of Texas by D. M. McLean and H. M. Meyer; and from the Imperial Valley by Robert Tang.

frozen for later study. The remainder of each shipped specimen as well as those grown in the greenhouse at Beltsville were prepared for inoculation by grinding the tissue with mortar and pestle in a 0.5% phosphate buffer (pH 7.8) solution. Inoculations were made to diagnostic hosts lightly dusted with carborundum using sterilized cheesecloth pads.

Diagnostic hosts used were Capsicum annuum (pepper cv. California Wonder), Vigna sinensis (cowpea cv. Blackeye), Nicotiana glutinosa, N. tabacum (tobacco cv. Samsun), Citrullus vulgaris (watermelon cv. Charleston Grey), Cucurbita pepo (squash cv. Early Prolific Straightneck), Cucumis melo var. cantalupensis (cantaloup cv. PMR 45), C. sativus (cucumber cv. Marketer), and Chenopodium amaranticolor.

The reactions of the test plants indicated the virus or viruses present in each specimen as follows: as a strain of CMV from local lesions produced on primary leaves of Blackeye southern pea, systemic mottle in pepper, tobacco and N. glutinosa, very severe systemic mottle in cantaloup, cucumber and squash, and necrotic spotting and severe stunting of inoculated leaves of watermelon and local lesions in 3 to 4 days on inoculated leaves of C. amaranticolor; SMV from veinbanding and interveinal blanching mottle on cantaloup and cucumber, moderate to severe mottle frequently accompanied with green blisters and leaf distortions in squash and no reaction on pepper, N. glutinosa, tobacco, watermelon, blackeye cowpea, and C. amaranticolor; TRSV from necrotic ring patterns on Samsun, greyish-white local lesions on cotyledons of squash (11), chlorotic stippling and mottle of secondary leaves of squash, cantaloup, cucumber, and watermelon, local lesions and top necrosis with subsequent death of most inoculated plants of blackeye cowpea and C. amaranticolor, no reaction on pepper and usually a mild reaction or none on N. glutinosa; WMV from a moderate to severe mottle, leaf distortion and stunting in cantaloup and watermelon, a moderate to severe mottle with green blisters in squash, a mild mottle in cucumber and no reaction on pepper, blackeye cowpea, tobacco, N. glutinosa and C. amaranticolor.

Viruses occurring in mixtures were separated when necessary for identification and further study as follows: CMV, SMV, and WMV from TRSV through inoculation of squash cotyledons (11), CMV from other viruses through pepper, WMV from SMV and CMV through watermelon, SMV from WMV and CMV through a dilution series (6) and TRSV from the other three viruses through systemic invasion of C. amaranticolor and blackeye cowpea.

The cucurbit hosts were inoculated in the cotyledon stage and other hosts in the 2 to 4 true-leaf stage.

Unless otherwise stated, greenhouse temperatures were maintained between 65° and 95° F. An insecticide was used twice weekly to insure freedom from insects. Weekly applications of a fungicide gave adequate control of powdery mildew.

RESULTS AND DISCUSSION

Locations and numbers of collections, hosts, number of isolates, and viruses present in the specimens are listed in Table 1. Of the 50 isolates from Crystal City, Texas, all were infected with TRSV and 18 were also infected with WMV. No other virus was detected in these specimens.

Table 1. Viruses identified in cucurbit specimens from Arizona, California, and Texas.

Location	Host	No. test locations	No. of isolates	Frequency of recovery of TRSV	WMV	SMV	CMV
Crystal City, Texas [a]	cantaloup	1	50	50	18	0	0
Lower Rio Grande Valley [b]	cantaloup	9	76	67	40	1	9
	cucumber	2	2	2	0	0	0
	honeydew	1	14	0	3	3	0
	squash	4	9	6	1	2	0
	watermelon	2	4	3	4	0	0
Mesa, Arizona [b]	cantaloup	1	40	0	27	7	21
Imperial Valley [b] (El Centro, California)	cantaloup	1	15	0	15	0	5
	squash	1	15	0	15	1	3
	watermelon	1	15	0	15	0	0

[a] Specimens received from both spring and fall plantings.
[b] Specimens from spring plantings only.

Of the 105 isolates from 18 widely separated locations in the Lower Rio Grande Valley, 78 were infected with TRSV, 48 with WMV, 6 with SMV, and 9 with CMV. Apparently the TRSV virus is the most important cucurbit virus but it is followed relatively closely by WMV. Specimens from only one field in the Rio Grande Valley were not infected with TRSV. SMV and CMV appear to be of little importance to melon production in southwest Texas (7).

Cantaloup specimens from Mesa, Arizona were infected predominantly with WMV and CMV (Table 1). Only 7 of the 40 samples were infected with SMV. Of the 45 specimens from the one location in the Imperial Valley of California, all were infected with WMV but, only 8 of the 30 squash and cantaloup specimens were infected with CMV and 1 with SMV (Table 1). Only one location each was sampled in Arizona and southern California and WMV was the virus most prevalent in both locations. CMV was almost as prevalent at Mesa as WMV; TRSV was not detected in isolations from Arizona or California specimens.

The results of these and other (4, 5, 7, 8) isolation studies indicate that viruses are widespread in the cucurbit-producing areas of Arizona, southern California, and southwest Texas. Although the major viruses in cucurbits apparently differ between the southern Texas and Arizona and California areas, most plantings are uniformly diseased well before harvest both in the spring crop as well as cucurbits planted in the fall in southern Texas. Although virus infection seriously reduces plant growth and marketable fruit production of the cucurbits, the presence of virus or viruses in melon plantings is not considered to be the sole cause of "crown blight" (8). However, tests under greenhouse conditions at Beltsville, Maryland indicate that the crown-blight syndrome can be induced in part in PMR 45 cantaloup through early virus infection and abnormally high temperatures before and during the melon-ripening period.

Literature Cited

1. ANDERSON, C. W. 1952. The distribution of cucurbit viruses in central Florida. Plant Disease Reptr. 36: 377-379.
2. DOOLITTLE, S. P. 1920. The mosaic disease of cucurbits. U. S. Dept. of Agr. Bull. 879, 69 pp.
3. FREITAG, J. H. 1952. Seven virus diseases of cucurbits in California. (Abst.) Phytopathology 42: 8.
4. GROGAN, R. G., D. H. HALL, and K. A. KIMBLE. 1959. Cucurbit mosaic viruses in California. Phytopathology 49: 366-376.
5. KEENER, P. D. 1958. Melon virus isolations and anatomical studies. Arizona Agr. Exp. Sta. Rept. No. 180: 13-14.
6. LINDBERG, G. D., D. H. HALL, and J. C. WALKER. 1956. A study of melon and squash mosaic viruses. Phytopathology 46: 489-495.
7. McLEAN, D. M., and H. M. MEYER. 1961. A survey of cucurbit viruses in the lower Rio Grande Valley of Texas: preliminary report. Plant Disease Reptr. 45: 137-139.
8. NELSON, M. R. 1961. Mosaic viruses not sole cause of crown blight. Progress Agriculture in Arizona 13: 13.
9. SHEPHERD, ROBERT J., and F. BEN STRUBLE. 1956. Tobacco ringspot virus on watermelon. Phytopathology 46: 358-362.
10. SINCLAIR, J. B., and J. C. WALKER. 1956. A survey of ringspot on cucumber in Wisconsin. Plant Disease Reptr. 40: 19-20.
11. WEBB, R. E. 1961. Separation and identification of some cucurbit viruses. Phytopathology Note (In press).

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE,
UNITED STATES DEPARTMENT OF AGRICULTURE

AN OUTBREAK OF CRONARTIUM COMPTONIAE ON MONTEREY AND BISHOP PINES ON VANCOUVER ISLAND, BRITISH COLUMBIA[1]

A. C. Molnar[2]

Abstract

A severe outbreak of infection by Cronartium comptoniae Arth. on Bishop pine (Pinus muricata) and Monterey pine (Pinus radiata) is reported in exotic forest plantations on Vancouver Island, British Columbia. Ninety-five percent of 5-year-old seedlings were infected in one plantation. The need for extra precautions to prevent introduction of C. comptoniae into regions of the world where Bishop and Monterey pines are economically important is emphasized.

INTRODUCTION

Forest tree species introduced from various parts of the world have been planted in British Columbia on a trial basis for several decades. Such experimental plantings have increased greatly since 1954, when the Forest Entomology and Pathology Laboratory, Canadian Department of Forestry, and the British Columbia Forest Service undertook their registration and surveillance. In the past 7 years the total number of plantations registered has increased from 30 to over 200, half of this total being made up of various species and varieties of pine. Plantations vary in size from less than 1 acre to 60 acres. A number of plantations have failed from different environmental causes, and insects and fungi are becoming increasingly evident as important agents of damage. This paper reports an outbreak of the sweet fern blister rust Cronartium comptoniae Arth. on Bishop pine (Pinus muricata) and Monterey pine (Pinus radiata).

FIELD OBSERVATIONS

Cronartium comptoniae is a native rust fungus that has been recorded relatively infrequently in British Columbia but occurs over a wide area of the Province. Until 1961 it was known in British Columbia only on lodgepole pine (Pinus contorta), its interior variety P. contorta var. latifolia, and Myrica gale, its known alternate host in British Columbia. A summary of its host range and distribution in North America is given by Boyce[3]. This summary does not include Pinus muricata or P. radiata and a further search of the literature has not revealed any record of the rust on these hosts.

In its aecial state on pine Cronartium comptoniae is difficult to distinguish from Peridermium stalactiforme Arth. & Kern. Positive identification is possible only by infection of the alternate host with aeciospores. Infections are easily overlooked except in the spring, when the aecial state is conspicuous on pine. In May 1961 C. comptoniae was found at high infection levels on test plantations of Monterey and Bishop pines on Vancouver Island. The identity of the fungus was confirmed by cross inoculations which gave positive results on Myrica gale and negative results on Castilleja sp.

The seriousness of the disease was demonstrated both by the number of infected plantations as well as by the intensity of infection in several areas (Table 1). Mortality of pine attributed to the rust comprised less than 1% of the infected stems in all areas examined but, because a high proportion of the infections involved the stem, heavy mortality may be expected in the future.

In the six planting areas where these pines occurred together Bishop pine always suffered a lower incidence of infection but, because it attained an infection level of 75% in one area, this apparently greater resistance to infection would not have any practical significance. The apparent resistance to infection of cluster pine (Pinus pinaster) was considerably more striking. This pine was present in 8 of the 10 plantations in which infected Monterey pine was found and in all cases it was free from infection by Cronartium comptoniae. It is, however, recorded as a host of this rust[3]. In another plantation where Monterey pine was 83% infected Austrian pine (Pinus nigra), cluster pine, and the hybrids P. echinata x taeda, P. murrayana x banksiana, and P. rigida x taeda were free from infection.

---

[1]Contribution No. 798 from the Forest Entomology and Pathology Branch, Department of Forestry, Ottawa, Canada.

[2]Forest Entomology and Pathology Laboratory, Victoria, B. C.

[3]Boyce, J. S. 1943. Host relationships and distribution of conifer rusts in the United States and Canada. Trans. Conn. Acad. Arts Sci. 35: 331-382.

Table 1. Plantations of Monterey and Bishop pines infected by Cronartium comptoniae on Vancouver Island.

Host	Total plantations examined	No. of plantations in infection classes (% of pine infected)				
		0	1-10	11-50	51-75	76-100
Monterey pine	12 (9000 trees)	2	5	1	1	3
Bishop pine	8 (3000 trees)	3	2	2	1	0

DISCUSSION

Only rarely does a forest tree species planted outside its natural range surpass the performance of an indigenous species. Monterey pine has attained this distinction in several parts of the world, but its susceptibility to Cronartium comptoniae casts doubt on its future success in British Columbia, at least in the coastal region. Bishop pine must also be regarded as a high risk tree in this region. These two new host records for C. comptoniae should caution against further plantings of the pines in British Columbia, at least until more is known of the distribution of the alternate host and other factors influencing severe infection. More important, however, is the need to guard against the introduction of this rust into regions of the world where Bishop or Monterey pines comprise an important part of the forest economy.

FOREST ENTOMOLOGY AND PATHOLOGY LABORATORY,
VICTORIA, BRITISH COLUMBIA

SPREAD AND DISTRIBUTION OF CAMELLIA FLOWER BLIGHT IN LOUISIANA

D. M. Alford, J. B. Sinclair and T. P. Pirone[1]

INTRODUCTION

Flower blight of camellias, incited by Sclerotinia camelliae Hara, was first described from Japan by Hara in 1919 (8). In 1940, Hansen and Thomas reported the disease in California (7). In 1943, Dr. Gilbert L. Stout of the California Department of Agriculture wrote to W. E. Anderson, State Entomologist of Louisiana[2]:

"The disease (camellia flower blight) was then (1941) known on only one property in California and it was believed at that time by those who worked on the disease, that it would thrive only under conditions of high humidity and under lath and that there would be little danger of it becoming established on plants grown without mulching in the open in parks and private gardens.

"I am writing you at this time to inform you that we have since found that the disease can occur in parks and private gardens. During the 1943 season we had a very wet spring and the disease appeared abundantly on camellias in parks and private yards in Sacramento and some other localities. Apparently it will be a factor in camellia flower production over a wider range of environmental conditions than was at first thought."

The first report of the disease outside of California came from Georgia in 1948 (6). The flower blight organism was believed to have been brought into that State on camellia plants brought in from California. By 1958 the blight fungus was known to be distributed in a number of southern States including Georgia (6), Louisiana (9), North Carolina (11), South Carolina (5), Texas (10), and Virginia (5).

SPREAD AND DISTRIBUTION OF CAMELLIA FLOWER BLIGHT IN LOUISIANA

Camellia flower blight was first reported from Louisiana in 1950 (3, 9). At that time it was found in the cities of Shreveport and Monroe in Caddo and Ouachita parishes, respectively (Fig. 1). In 1951 the disease was found in New Orleans in Orleans Parish (3). The introduction of camellia

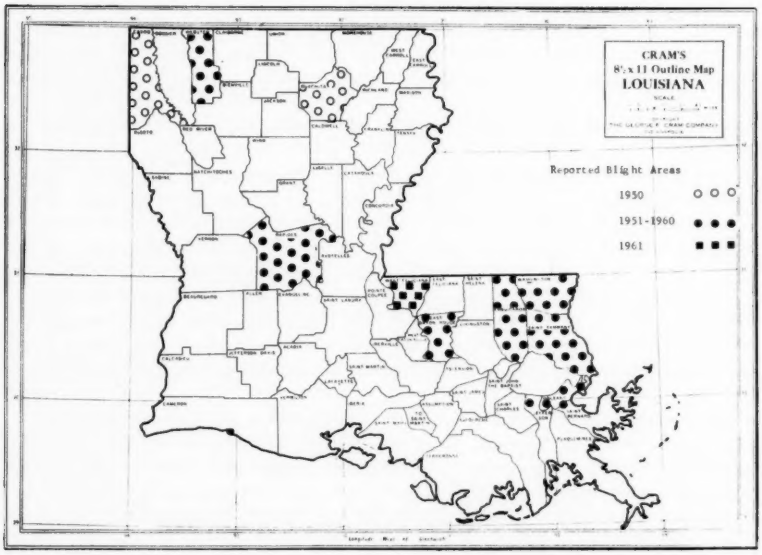


FIGURE 1. Distribution in Louisiana, by parish, of reported sites infested by the camellia flower blight organism from 1950-1961.

[1]Instructor of Biology, McNeese State College; Associate Professor of Plant Pathology, Louisiana State University; and Assistant Professor of Plant Pathology, Louisiana State University, respectively.

[2]From a letter dated November 24, 1943 to Dr. C. W. Edgerton from W. E. Anderson. In the files of Louisiana State University Department of Plant Pathology, Baton Rouge.

flower blight into Louisiana was traced back to infected plants brought into the State from California (2). Although quarantine and eradication measures were carried out in Louisiana in an attempt to limit the spread of the disease or to eradicate it from these originally infested areas, camellia flower blight spread rapidly to many new properties in these areas. In New Orleans there were 133 properties known to be infested in 1956 and this number increased to 859 in 1957 (3). By 1960, the disease had been found in the following Louisiana parishes: Caddo, East Baton Rouge, Jefferson, Orleans, Ouachita, Rapides, Tangipahoa, Washington, Webster (12) and St. Tammany. In Rapides Parish only a single infected bloom was found in a camellia flower display. However, the infected flower may have come from New Orleans rather than from the immediate area. Rapides has not been confirmed as an infested parish.

During the 1960-61 camellia season a more thorough survey of camellia petal blight in Louisiana was made with the cooperation of the State Department of Entomology. An area or planting was not considered infested unless an apothecium from a germinating sclerotium was found on the property being surveyed and/or laboratory diagnosis of diseased flowers was made (1).

The known areas of infestation were found to have spread since 1960. Mr. Edwin Lott, State Entomologist, reported that the entire greater New Orleans area could now be considered infested[3]. All of the Covington area in St. Tammany Parish was considered infected after the 1961 survey including an area 5 miles north of the Covington city limits and south to the city of Mandeville. The number of infested sites in East Baton Rouge Parish have enlarged to include all of the southern section of the city of Baton Rouge, the Louisiana State University Horticulture Garden camellia plantings, as well as areas south of the city limits. West Feliciana was added to the list of parishes with known areas of flower blight infestation. Two large plantings in this parish had infected flowers and sclerotia producing apothecia. Apparently the area east of the Mississippi River in Louisiana is the area of most rapid dissemination of the disease, and if the disease continues to spread at the present rate this entire area soon can be considered infested.

Continued enforcement of quarantine regulations and the use of recommended ground sprays (3, 12) are important in keeping the disease from spreading to other areas in the State.

Literature Cited

1. ALFORD, D. M. 1961. Studies on camellia flower blight. Unpublished M. S. Thesis, Louisiana State University Library, Baton Rouge.
2. ANZALONE, L., Jr. 1959. Studies on flower blight of camellia and Cercospora leaf spot of Photinia serrulata. Unpublished Ph.D. Dissertation, Louisiana State University Library, Baton Rouge.
3. ANZALONE, L., Jr., and A. G. PLAKIDAS. 1958. Control of flower blight of camellias in Louisiana with fungicides. Plant Disease Reptr. 42: 804-806.
4. BAXTER, L. W., and W. M. EPPS. 1958. Camellia flower blight. Clemson, South Carolina Mimeo Series #5. [not cited in text]
5. BROWN, A. C. 1954. Camellia flower blight. American Camellia Yearbook 34: 40.
6. GILL, D. L. 1948. Camellia flower blight in the South. Plant Disease Reptr. 32: 317-318.
7. HANSEN, H. H., and E. THOMAS. 1940. Flower blight of camellias. Phytopathology 30: 166-170.
8. HARA, K. 1919. On a Sclerotinia disease of camellia (In Japanese). Dainippon Saurinkwaiko 436: 29-31.
9. PLAKIDAS, A. G. 1950. Camellia flower blight in Louisiana. Plant Disease Reptr. 34: 188.
10. PLAKIDAS, A. G. 1957. Camellia petal blight found in Texas. Plant Disease Reptr. 41: 547.
11. WINSTEAD, N. N., E. C. NELSON, and F. A. HAASIS. 1954. Distribution of Sclerotinia camelliae in North Carolina, 1950-1954. Plant Disease Reptr. 38: 670-672.
12. ZUMMO, N. 1960. Studies on chemical control of petal blight of camellia incited by Sclerotinia camelliae. Unpublished Ph.D. Dissertation, Louisiana State University Library, Baton Rouge.

McNEESE STATE COLLEGE, LAKE CHARLES AND LOUISIANA STATE UNIVERSITY, BATON ROUGE, LOUISIANA

[3]Lott, E. 1961. Personal communication.

CONTROL OF PYTHIUM ROOT DISEASES WITH SOIL FUNGICIDES[1]

J. Tammen, D. P. Muse, and J. H. Haas[2]

Summary

Captan, ferbam, thiram, zineb, and certain experimental chemicals were mixed with steam treated soils and compared to determine relative effectiveness in limiting colonization by Pythium spinosum for an extended time. The fact that thiram persists in soil for only a short time was verified. Captan persisted in soil for 92 days, but its expressed activity during this period was directly dependent upon the environment as it affected disease proneness of the suscept and/or infection potential of the fungus. Dexon limited colonization for the 92-day period at concentrations not phytotoxic and its expressed activity remained at a high level independent of the environment.

Root diseases incited by Pythium represent one of the limiting factors in the production of floricultural crops in Pennsylvania. Significant losses are experienced annually in the production of pot plants, bedding plants, and cut flowers. In addition to causing direct losses, the fungus may infect roots without killing the plant, effecting the indirect losses of reduced quality and increased costs.

The pathogen is a soil inhabitant, occurring in most agricultural soils (4). When such soils are utilized for intensive greenhouse crop production, losses can be expected unless the soil is treated. The most widely used soil treatment in Pennsylvania greenhouses to eradicate soil-borne plant pathogens is free flowing steam. Though the suggested steam treatment is 180° F for 30 minutes, because of the nature of free flowing steam, much of the soil is treated at 212° F for periods exceeding 30 minutes. This serves not only to eradicate species of Pythium, but also a large part of the saprophytic soil microflora. Due to the absence of competition and/or antagonism by the saprophytic microflora, Pythium, when re-introduced, rapidly colonizes the soil mass, causing losses which may exceed those encountered in soils not treated with steam. As a result, it has been found necessary to establish a rigorous sanitation program in conjunction with steam treatment (3).

Sanitation in the commercial production of floricultural crops is an ideal rather than an accomplished fact. Widespread losses continue to occur, and additional means must be found to limit colonization of steam treated soils by plant pathogens. Replacing steam with chemical fumigation is not entirely satisfactory because of toxicity problems encountered under glass and because of increased costs. The use of low temperature, aerated steam that would destroy Pythium but permit survival of its competitors and/or antagonists would be a logical solution (1).

The use of soil fungicides or soil fungistats in conjunction with the recommended steam treatment -- to prevent reinfestation -- has promise as a practical solution to the problem. Although few, if any, currently available fungicides may be used to eradicate plant pathogenic fungi from soil unless used at concentrations toxic to plant growth, their use to limit colonization or to appreciably reduce damping-off has been demonstrated. Thiram has been reported as exhibiting a high initial level of activity against Pythium, with activity decreasing rapidly with time (2, 5, 7). Similar results have been achieved with zineb and captan (5, 7), but reports concerning activity over time, particularly with captan, vary. Thus, Scheffer and Haney (8) state that the protection afforded by captan (225 ppm) was lost within 35 days. Kennedy and Brinkerhoff (5) found that the fungicide (50 ppm) was as effective after 28 days in the soil as it was initially if testing occurred in the summer, but that in the winter when temperatures retarded emergence of the test plant (cotton) the chemical had little effect. Munnecke (6), using Myrothecium verrucaria (Alb. & Schwein.) Ditm. ex Fr. as a test organism, found that captan (667 ppm) added to steam treated, propylene oxide treated or non-treated soil exhibited little or no loss in activity for 63 days, the period of his test. Domsch (2) states that the duration of activity of captan mixed with soil is related to the concentration; the smaller the amount added, the

---

[1]Contribution No. 280 of the Department of Botany and Plant Pathology, Pennsylvania Agricultural Experiment Station. Authorized for publication as paper No. 2578 in the Journal Series of the Pennsylvania Agricultural Experiment Station.

[2]Assistant Professor of Plant Pathology, Graduate Student in Horticulture, and Research Assistant in Plant Pathology, respectively.

earlier the loss of activity. He found that at 250 ppm the half life was 70 to 75 days; at 100 ppm, 50 days; and at 50 ppm, 20 to 30 days.

An ideal fungicide would at least be non-toxic to growing plants, effective in preventing the establishment of Pythium over the period of time required to produce a crop, and effective under conditions most favorable for development of the pathogen. Tests to identify such a fungicide have been the subject of study by the authors for 3 years. Initial experiments were designed to evaluate the effectiveness of selected currently marketed fungicides. The most effective of these were compared with recently developed chemical compounds.

METHODS

The experiments were carried out in the greenhouse at a minimum temperature of 75° F. Standard 6-inch pots were filled with soil (1:1:1 mixture of Hagerstown silt loam, sphagnum peat and river bottom sand). During the process of filling, an inverted 18 mm o.d. test tube was placed at the center of the pot to provide a vacant cylinder from top to bottom for the eventual introduction of inoculum (Fig. 1a). The pots were then placed on copper naphthenate treated wood blocks on the greenhouse bench, covered with a plastic sheet and steam treated at 212° F for 30 minutes. After the soil had cooled, fungicides were added at the desired concentrations, calculated as ppm active ingredient by soil volume, and the soil and fungicide thoroughly mixed.

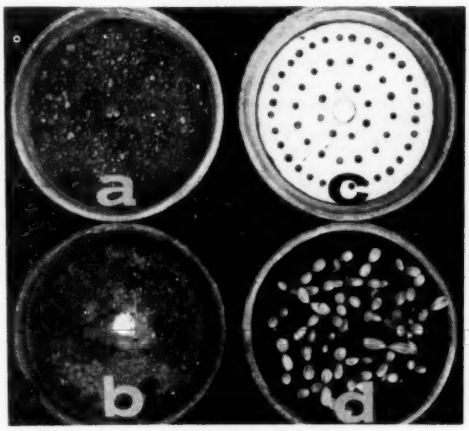


FIGURE 1. Fungicide screening: a -- Pot with test tube. b -- Test tube removed, inoculum introduced. c -- Planting guide, seedlings planted in three rows about the central core of inoculum. d -- Germinated zinnia seedlings.

Infestation of the soil was accomplished by removing the test tube and introducing 7 day, sand-corn meal (1% corn meal, 99% washed quartz sand, by weight) cultures of Pythium spinosum Sawada[3] into the central hole to within 1/2 inch of the soil surface (Fig. 1b).

Zinnia (Zinnia elegans 'Purity') was selected for use as a test plant because the seeds germinate rapidly (3 to 5 days) and the emerging seedlings are highly susceptible to attack by the pathogen. Seeds were planted in three concentric circles about the center of the pot (Fig. 1c and 1d). Damping-off of seedlings from the center toward the rim of the pot served as a measure of the efficiency of a fungicide in limiting colonization by the pathogen. Duration of activity was measured from the date of mixing a fungicide with the soil. Except where otherwise noted, four pots were used for each treatment and each treatment was replicated four times.

EXPERIMENTAL RESULTS

Currently Marketed Fungicides: Selected chemicals, 76% ferbam, 65% zineb, 75% thiram, 50% captan, and 50% N-trichloromethylthiophthalimide (Phaltan) were tested at three concentrations approximating the recommended, one-half the recommended, and twice the amounts recommended by their manufacturers.

To determine if the chemicals could be mixed with the soil prior to the steam treatment, a series identical to the above was prepared and steam treated at 212° F for 30 minutes. Both series were planted a second time, using the same pots to obtain an indication of duration of activity. Data were collected at 8 and 15 days after mixing. The results are summarized in Table 1.

At the end of the 8-day period, only ferbam at 240 ppm (active ingredient) prevented colonization. Thiram at all concentrations and Phaltan at 40 ppm (active ingredient) limited colonization, but not significantly. Phaltan at the lowest concentration (40 ppm active ingredient) was more efficient than at the two higher concentrations, indicating possible phytotoxicity. Zineb and captan were ineffective. Increasing the concentration of each chemical did not result in increased efficiency, except with ferbam.

[3]Originally isolated from the basal stem of Chrysanthemum morifolium and tentatively identified to species by Dr. J. T. Middleton, Department of Plant Pathology, University of California, Riverside.

Table 1. Efficiency of various fungicides mixed with soil in limiting colonization by Pythium spinosum.

Treatment	Concentration (ppm active ingredient)	Number of days after mixing: 8		15	
		not steamed	steamed[a]	not steamed	steamed[a]
Ferbam	60	[b]42.3[c]	[b]48.0	[b]18.0[d]	[b]23.5
	120	44.5	44.3	27.5	23.3
	240	49.5	44.0	27.0	27.5
Zineb	50	37.8	39.5	18.0	19.8
	100	42.3	44.3	21.5	21.0
	200	41.3	47.0	22.8	24.5
Thiram	50	47.3	43.5	17.0	25.0
	100	46.5	43.3	26.5	22.3
	200	45.5	37.5	40.3	22.8
Captan	40	43.3	44.0	32.0	29.5
	80	41.8	41.5	31.0	16.5
	120	42.3	39.8	40.8	21.8
Phaltan	40	46.0	24.5	34.8	17.8
	80	39.0	29.5	24.8	26.8
	120	35.8	17.0	40.0	22.8
Infested control	---	41.8	41.8	20.0	20.0
Control	---	51.0	51.0	42.3	42.3

[a]Fungicides added and steam treated at 212° F for 30 minutes.
[b]Number of seedlings surviving of 56 planted per pot; average of 16 pots.
[c]$t_{.01} = 8.6$; $t_{.05} = 6.4$ [d]$t_{.01} = 15.2$; $t_{.05} = 11.3$.

Table 2. Efficiency of experimental fungicides and thiram in limiting colonization by Pythium spinosum.

Treatment	Concentration (ppm active ingredient)	Number of days after mixing: 9	19	26	36
Thiram	50	44.8[a]	17.5[d]	6.5[e]	2.5[f]
	100	46.8[c]	30.2	10.5	2.0
	200	48.3	40.5	22.8	10.3
B-1843	140	---[b]	39.8	42.5	41.5
	210	---	37.0	38.0	34.0
	280	---	36.0	42.3	36.8
D-113	95	---[b]	32.7	22.5	3.5
	195	---	39.0	40.8	17.3
	284	---	33.8	36.8	22.5
Dexon	50	44.3	43.0	43.3	39.5
	95	48.9	44.3	41.0	41.3
	142	48.0	46.8	42.8	41.0
CP 15986	140	---[b]	47.0	38.5	39.3
	280	---	46.5	37.8	44.0
	420	---	44.5	40.5	42.8
CP 30249	70	---[b]	35.8	28.3	31.8
	140	---	36.5	30.5	39.8
	210	---	34.3	28.8	23.8
Infested control	---	41.3	2.0	2.8	0.5
Control	---	49.5	45.3	49.8	35.5

[a]Number of seedlings surviving of 56 planted per pot; average of 16 pots.
[b]All levels phytotoxic at 9 days; data not recorded.
[c]$t_{.01} = 5.7$; $t_{.05} = 4.2$ [d]$t_{.01} = 13.8$; $t_{.05} = 10.2$ [e]$t_{.01} = 12.7$; $t_{.05} = 9.4$ [f]$t_{.01} = 13.0$; $t_{.05} = 9.6$

At the end of 15 days, ferbam exhibited a marked reduction in activity. Increased efficiency with increased concentration, not apparent at 8 days, became evident with thiram and Phaltan, but these chemicals limited colonization only at the highest levels tested. Captan, ineffective at 8 days, effectively limited colonization at the highest level tested.

Steam treatment had no effect on the activity of ferbam, zineb or captan, but resulted in a highly significant reduction in the activity of thiram and Phaltan.

Thiram Versus Experimental Chemicals: Experimental chemicals trans-1, 2-bis (n-propylsulfonyl) ethylene (Chemagro B-1843), 1, 2-dichloro-1-(methylsulfonyl) ethylene (Chemagro D-113), p-dimethylaminobenzenediazo sodium sulfonate (Dexon), (sodium methyoxyethyldithiocarbamate (Monsanto CP 15986), and 2-chloro-3(tolylsulfonyl)-propionitrile (Monsanto CP 30249) were compared with thiram. As in the previous experiment, each of these was tested at three concentrations. CP 15986 (50% aqueous soln) and CP 30249 (4 pounds/gallon) are liquids and the desired amount of these was applied in enough water to wet the soil to a point above field capacity, but below container capacity. D-113, B-1843, and Dexon were derived from 5% granular formulations and thiram from the 75% wettable powder.

To determine duration of activity, the test period was extended by planting zinnia seeds in the same test pots at 9, 19, 26, and 36 days after adding the chemicals to the soil. The results are reported in Table 2.

Although thiram was initially effective, the highly significant reduction in stand at 19, 26, and 36 days shows that it would not serve to limit colonization over an extended period of time. The same applies to Chemagro D-113.

The remaining chemicals exhibited a high degree of activity in comparison to thiram, limiting colonization by Pythium during the entire test period. The initial phytotoxicity of Chemagro B-1843, Monsanto CP 15986, and Monsanto CP 30249 would limit use at the tested concentrations. Phytotoxicity of these chemicals was also evident at 19 days, but was not severe enough to prevent recording of data. Dexon proved outstanding; it effectively limited colonization for the 36-day period at the lowest concentration and was non-phytotoxic at the highest concentration.

Captan Versus Dexon: The period of this experiment was extended to 92 days. To avoid contamination during this time, both the infested and non-infested control were freshly prepared at the beginning of each test period. The chemicals, however, were mixed with the steam treated soil from each pot at the stated concentrations at the beginning of the test period and the pots containing the mixtures were stored on the greenhouse bench until the desired planting dates.

At these times, infestation and planting was carried out as previously described except that the number of seeds planted was reduced from 56 to 40 per container. Each treatment was represented by three pots and was replicated four times. Time was calculated from date of mixing. Captan was used as the 50% wettable powder and Dexon as the 5% granular formulation.

Dexon, at 50 ppm active ingredient, was highly effective in limiting colonization of the soil by Pythium during the entire test period and was significantly more effective than captan at the same concentration (Fig. 2).

The activity of both chemicals varied with time. Thus, though stands achieved through the use of Dexon did not differ from those of the noninfested control at 18, 35, 50, 57 or 92 days, a significant difference occurred at 64 days. Though stands achieved with the use of captan did not differ significantly from those of the infested control at 18, 35, 57 or 64 days, a significant difference occurred at 50 and 78 days. At 35 days, captan exhibited no apparent activity, but at 50 days the stand achieved with this chemical did not differ significantly from that of the noninfested control.

It is difficult to ascribe this variability in activity to any single factor. Soil moisture and temperature were major variables between experiments. Temperature was conditioned by thermostatically controlled heating and cooling to 75° F, but temperatures ranged to 95° F on sunny days. No attempt was made to control moisture and observations indicate that this was the primary variable factor.

DISCUSSION

The results of the experiments with thiram compare favorably with similar tests previously reported (2, 5, 7). The activity of this fungicide when mixed with soil rapidly declines and it would not serve to limit colonization of steam treated soil by Pythium for the period required. The same applies to the use of zineb, ferbam, and Chemagro D-113.

An accurate assessment of the efficiency of Chemagro B-1843, Monsanto CP 15986, and Monsanto CP 30249 can not be made because of the initial toxicity encountered at the concentrations tested. The results with these, particularly with the latter two, easily applied,

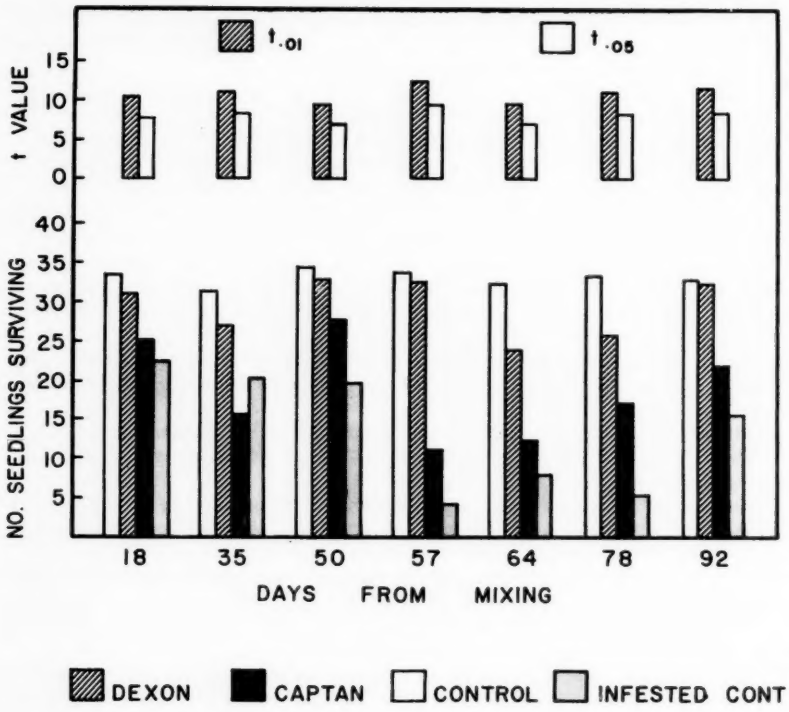


FIGURE 2. Efficiency of Dexon compared with that of captan in limiting colonization of soil by Pythium spinosum.

liquid compounds, would indicate value in testing at lower concentrations.

Captan remains active for long periods of time when mixed with soil. Results of the captan versus Dexon tests showed that captan (50 ppm active ingredient) retained its initial level of activity for 92 days. Because of differing concentrations and experimental procedures, it is difficult to make direct correlations between these results and those reported earlier (2, 5, 6, 8). It would seem, however, that the duration of activity of captan in soil is linked to factors other than initial concentration and that these factors may completely override any concentration effect. Kennedy and Brinkerhoff (5) relate difference in activity to the effect of temperature on the suscept. It is suggested here that soil moisture is a primary factor. This indicates that the "intrinsic" activity of captan had not changed, but that the expressed activity changed as the environment altered the disease proneness of the suscept and the infection potential of the fungus.

Of the materials tested, Dexon most satisfactorily meets the requirements of a fungicide to limit colonization of steam treated soil by Pythium. Not only does this chemical retain its initial level of activity over an extended period of time, but its expressed activity remains at a high level independent of the environment.

Literature Cited

1. BAKER, KENNETH F., and CHESTER N. ROISTACHER. 1957. Principles of heat treatment of soil. In the U. C. System for producing healthy container grown plants. California Agr. Exp. Sta. Ext. Serv. Manual 23. p. 332 (148).
2. DOMSCH, K. H. 1958. Die Wirkung von Bodenfungiciden. Z. Pflkrankh. 65: 651-656.
3. FERGUSON, JOHN. 1957. Beneficial soil microorganisms. In the U. C. System for producing healthy container grown plants. California Agr. Exp. Sta. Ext. Serv. Manual 23. p. 332 (237-254).
4. GARRETT, S. D. 1956. Biology of root-infecting fungi. Cambridge University Press, Cambridge. p. 293 (45-52).

5. KENNEDY, B. W., and L. A. BRINKERHOFF. 1959. Comparison of four soil fungicides in the greenhouse for the control of seedling diseases of cotton. Plant Disease Reptr. 43: 90-97.
6. MUNNECKE, DONALD E. 1958. The persistence of nonvolatile diffusible fungicides in soil. Phytopathology 48: 581-585.
7. RICHARDSON, L. T. 1954. The persistence of thiram in soil and its relationship to the microbiological balance and damping-off control. Can. J. Botany 32: 335-346.
8. SCHEFFER, R. P., and W. J. HANEY. 1956. Causes and control of root rot in Michigan greenhouses. Plant Disease Reptr. 40: 570-579.

PENNSYLVANIA AGRICULTURAL EXPERIMENT STATION, UNIVERSITY PARK

CONTROL OF PYTHIUM ROOT AND BASAL STEM ROT OF CHRYSANTHEMUM MORIFOLIUM WITH DEXON[1]

J. Tammen and D. F. Muse[2]

Summary

Greenhouse tests showed that Dexon, when mixed with the soil at 45 ppm (active ingredient), was highly effective in controlling Pythium root and basal stem rot of chrysanthemum. At 30 ppm, Dexon effected a highly significant but unsatisfactory degree of control; at 15 ppm, test plants differed little from infested, nontreated checks. Tests using noninfested steam treated soil to which Dexon had been ammended at 15, 30, and 45 ppm (active ingredient) indicated that the chemical was not toxic to chrysanthemum at these levels. It is concluded that Dexon combined with soil at 45 ppm (active ingredient) will control the disease.

Species of Pythium have long been known to cause a root and basal stem rot of greenhouse and field grown chrysanthemums. Tompkins and Middleton (5, 6) reported a serious outbreak of the disease on cuttings and rooted cuttings in the propagating bench in 1944 and 1950. Epiphytotics occurred in the major chrysanthemum-growing areas of Florida during 1955-1959 (1, 2, 3). Serious losses have occurred in Pennsylvania during the past 4 years, particularly with chrysanthemums grown in ground beds in poorly ventilated plastic greenhouses (4).

Symptoms resulting from attacks by the fungus apparently vary with temperature and moisture. Observations indicate that with high temperatures (80° to 90° F), high relative humidities, and high soil moisture -- conditions frequently encountered in Florida during the spring and fall or in Pennsylvania during the summer -- the first symptom is a wet, black-brown basal stem rot that may extend several inches above the soil surface. Affected plants rapidly wilt and collapse. Often the fungus invades leaves in contact with the soil and progresses into the stem at the node, causing a black-brown lesion 2 to 3 inches above the soil surface. In such cases the basal stem and roots may be essentially symptomless. Quick, but not always accurate, diagnosis may be made by mounting affected stem tissues in cotton blue and lactophenol, heating, and observing oospores and sporangia of the fungus. Under conditions of low temperature (50° to 65°) and high soil moisture affected plants are stunted and exhibit various nutrient deficiency symptoms; root systems are reduced in extent and are affected with a wet, brown to dark-brown rot. Aboveground basal stem lesions are seldom observed.

Control of the soil-borne diseases of chrysanthemum has depended primarily upon steam treatment or chemical fumigation of infested soil and sanitation to prevent introduction of the pathogens. Though these practices have been and are used with success in controlling Pythium root and basal stem rot of chrysanthemum grown in containers and raised or concrete V-bottom

[1]Contribution No. 281 of the Department of Botany and Plant Pathology, Pennsylvania Agricultural Experiment Station. Authorized for publication as Paper No. 2579 in the Journal Series of Pennsylvania Agricultural Experiment Station.
[2]Assistant Professor of Plant Pathology and Graduate Student in Horticulture, respectively.

ground benches, they often fail with production in ground beds because of the rapid re-colonization of treated soil by the pathogen. Fungicides or fungistats (such as ferbam, captan, zineb, Pano-drench, and others) have been widely used with varying success to limit colonization. Because of the demonstrated efficiency of p-dimethylaminobenzenediazo sodium sulfonate (Dexon) in limiting colonization of steam treated soil by Pythium (4), tests were designed to determine its effectiveness in controlling Pythium root and basal stem rot of chrysanthemum.

Four-inch clay pots were filled with equal volumes of soil (1:1:1 mixture of sphagnum peat, sand and silt loam), placed on individual copper naphthenate treated wood blocks, and steam treated at 212° F for approximately 30 minutes. After the soil had cooled, the desired amount of 5% granular Dexon, calculated as ppm active ingredient by soil volume, was thoroughly mixed with the soil from each pot. Pathogen-free cuttings of Chrysanthemum morifolium 'Iceberg' were rooted under aseptic conditions and planted one per pot.

Twelve days after planting, the soil was infested with 7-day-old sand-corn meal cultures[3] of Pythium spinosum Sawada[4] originally isolated from chrysanthemum. Infestation was accomplished by distributing 15 grams of the culture on the surface of the soil around the rim of the pot.

Three concentrations of Dexon, 15, 30, and 45 ppm (active ingredient), were compared with infested and noninfested controls. Each treatment was composed of four pots and each treatment was replicated four times. An identical noninfested series was set-up to determine the effect of Dexon on the growth of the plant.

The rooted cuttings were planted on April 12, 1960 and harvested on June 13, 1960 (9 weeks). The efficiency of the Dexon treatments was measured by determining the green weight of the tops of the plants and by rating the roots on the surface of the root ball for extent of the root system and severity of root rot symptoms. Extent of the root system was rated as the percentage of the outer ball occupied by roots as follows: Class 1.0, 100%; Class 2.0, 75%; Class 3.0, 50%; Class 4.0, 25%; Class 5.0, no roots. Severity of root rot was rated as the percentage of the rotted roots on the outer balls as follows: Class 1.0, no root rot; Class 2.0, 25%; Class 3.0, 50%; Class 4.0, 75%; Class 5.0, all roots rotted.

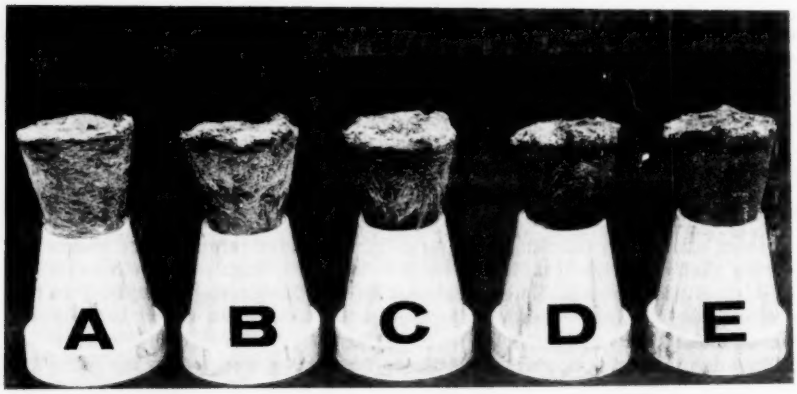


FIGURE 1. The influence of Dexon on development of Pythium root and basal stem rot of chrysanthemum. Root symptoms, left to right: A -- noninfested control, B -- 45 ppm Dexon, C -- 30 ppm Dexon, D -- 15 ppm Dexon, E -- infested control.

RESULTS

Dexon was highly effective in controlling the disease (Table 1, Fig. 1). With increasing concentrations of the chemical from 15 to 45 ppm, the severity of root rot decreased, the extent of the root system increased, and the green weight of the tops increased. At the lowest concentration, though green weight differed significantly, root rot severity and extent of the root system did not differ from the infested control. At the highest concentration, though green weight differed significantly, root rot severity and extent of the root system did not differ from the noninfested control. The chemical at 30 ppm effected a highly significant but unsatisfactory degree of control.

Dexon at the highest concentration tested caused neither visible nor measurable toxicity to chrysanthemum.

---

[3] 1% corn meal, 99% washed quartz sand by air dry weight.

[4] Tentatively identified to species by Dr. J. T. Middleton, Department of Plant Pathology, University of California, Riverside.

Table 1. Influence of Dexon mixed with soil on the development of Pythium root and basal stem rot of chrysanthemum.

Treatment	Concentration (ppm active)	Green weight (grams)	Root rot[a] severity	Extent[b] root system
Infested control	---	66[c]	3.9[d]	3.3[e]
Dexon	15	104	3.3	2.8
	30	125	2.3	2.1
	45	153	1.8	1.4
Noninfested control	---	184	1.0	1.0

[a]Based on scale of from 1.0 - 5.0; 1.0 = no root rot, 5.0 = all roots on outer surface of root ball rotted.

[b]Based on scale of from 1.0 - 5.0; 1.0 = entire outer surface of root ball occupied by roots, 5.0 = no roots on surface of outer ball.

[c]$t_{.01} = 39.1$; $t_{.05} = 29.5$. [d]$t_{.01} = 1.2$; $t_{.05} = 0.9$. [e]$t_{.01} = 0.8$; $t_{.05} = 0.6$.

DISCUSSION

The actual amount of Dexon that should be combined with the soil to effectively prevent the establishment of Pythium is difficult to ascertain. Though the inoculum levels utilized in the experiment were probably higher than those commonly experienced in nature and it may be valid to assume that concentrations of less than 45 ppm would be effective, no such conclusion can be drawn without data pertaining to the influence of inoculum potential and/or nutrient base on colonization of the soil and disease development. Further, the experimental data reported here would indicate that a higher degree of control might be achieved with concentrations exceeding 45 ppm. Until additional data are available, it is suggested that 45 ppm (active ingredient) of the 5% granular formulation of Dexon be used.

Literature Cited

1. COE, D. M. 1958. Some recent developments in diseases of ornamental plants in Florida. Proc. Florida Hort. Soc. for 1957, 70: 390.
2. JACKSON, C. R. 1959. Control of Pythium and Rhizoctonia in chrysanthemum plantings. University of Florida, Gulf Coast Exp. Sta. Mimeo. Rept. 60-3. Nov. 16.
3. TAMMEN, JAMES. 1957. Report of the Ornamentals Pathology Department, State Plant Board of Florida, 21st Biennial Report (July 1954 - June 1956), 78-89.
4. TAMMEN, JAMES, D. P. MUSE, and J. H. HAAS. 1961. Soil fungicides and the control of Pythium root diseases. Pennsylvania Flower Growers Bull. No. 121: 1, 10 - 11.
5. TOMPKINS, C. M. 1944. Basal rot of mums in California plantings. Florists' Review 94(2424): 23.
6. TOMPKINS, C. M., and JOHN T. MIDDLETON. 1950. Control of basal rot of cuttings of chrysanthemum and other ornamentals. Bull. Torrey Botan. Club 77: 287-293.

PENNSYLVANIA AGRICULTURAL EXPERIMENT STATION, UNIVERSITY PARK

A NON-FUNGISTATIC BACTERIOSTATIC MEDIUM CONTAINING STREPTOMYCIN AND VANCOMYCIN

A. J. Latham and M. B. Linn[1]

Summary

Potato-dextrose agar amended with streptomycin and vancomycin was used for plating out mycelial-agar disks recovered from soil columns drenched with fungicides. This medium was bacteriostatic and non-fungistatic in cultures of five soil fungus pathogens. It was superior in all instances to media containing rose bengal and in a few instances to a medium supplemented with lactic acid or novobiocin.

INTRODUCTION

Chemicals have been used to inhibit the growth of bacteria and fungi in culturing soil microflora. Waksman (7) added lactic acid to an agar medium to inhibit bacterial growth in soil dilution plates. Martin (3) used rose bengal and streptomycin sulfate in a peptone-dextrose medium to prevent bacterial growth and to retard the growth of fungi. Butler and Hine (1) amended potato-dextrose agar with novobiocin (Albamycin) to inhibit bacteria in isolating fungi from the soil.

Newhall (5) used Martin's medium and Cetas and Whidden (2) employed that of Butler and Hine in evaluating fungicides applied as drenches in soil columns (5). The fungus mycelial-agar disks were recovered from the soil after exposure to the fungicides and plated on the media to determine viability. Preliminary tests in our laboratory revealed that Martin's medium and potato-dextrose agar plus novobiocin did not permit normal growth of certain soil fungus pathogens.

Table 1. Growth of five fungi on media containing fungistatic or bacteriostatic compounds.

Media	Colony diameter (mm)[a]				
	Diplodia zeae (72)[b]	Pyrenochaeta terrestris (96)	Pythium debaryanum (24)	Rhizoctonia solani (48)	Sclerotium cepivorum (96)
PDA (Control)	83.3 a	30.3 a	54.6 a	71.0 a	67.3 a
PDASV	80.3 a	31.3 a	58.0 a	70.6 a	66.6 a
PDA + lactic acid	80.6 a	14.3 b	5.0 b	44.6 b	66.3 a
PDA + novobiocin	28.3	30.0 a	19.6	71.0 a	29.0
Martin's medium	13.6 b	10.6 b	8.0 b	38.3 b	10.0 b
PDA + rose bengal + streptomycin	10.0 b	12.0 b	8.0 b	16.6	9.0 b

[a] Diameters followed by the same letters in each vertical column are not significantly different by Duncan's Multiple Range Test; others are at the 1% level.
[b] Period of growth in hours.

This paper reports comparative tests with five bacteriostatic media containing one or more growth inhibiting compounds in the development of a non-fungistatic medium to five soil fungus pathogens.

MATERIALS AND METHODS

Five media having fungistatic or bacteriostatic properties were prepared: Martin's medium; potato-dextrose agar (PDA) + rose bengal (1:30, 000) + streptomycin sulfate (30 μg/ml); PDA + novobiocin (100 μg/ml); PDA + lactic acid (pH adjusted to 4.0); and PDA + streptomycin sulfate (100 μg/ml) + vancomycin (50 μg/ml) (PDASV). The streptomycin-vancomycin combination was selected because streptomycin inhibits gram-negative bacteria (6) and vancomycin primarily gram-positive bacteria (4).

[1] Research Assistant and Professor of Plant Pathology, respectively, Department of Plant Pathology, University of Illinois, Urbana.

The test pathogens were: Diplodia zeae (Schw.) Lév.; Pyrenochaeta terrestris (Hans.) Gorenz, J. C. Walker, & Larson; Pythium debaryanum Hesse; Rhizoctonia solani Kuehn; and Sclerotium cepivorum Berk. The inhibiting compounds were added to the PDA (Difco, pH 5.6) after it had cooled to 47°C. To determine growth inhibition of the five pathogens, a 5-mm mycelial-agar disk approximately 2-mm thick was cut from the advancing margin of a pure culture of each pathogen and placed in the center of each of three Petri plates containing the different media. The plates were incubated at room temperature (23° to 24°C) and growth recorded at 24-hour intervals.

RESULTS AND DISCUSSION

Potato-dextrose agar plus streptomycin and vancomycin (PDASV) was the most suitable bacteriostatic medium since it had no significant effect on growth of the five test fungi (Table 1). Lactic acid did not inhibit the growth of Diplodia zeae and Sclerotium cepivorum though it did that of Pythium debaryanum. Novobiocin did not suppress Pyrenochaeta terrestris and Rhizoctonia solani but it did P. debaryanum as previously reported (1). The addition of rose bengal to a medium had a marked fungistatic effect.

On the basis of these results, it would be unwise to use any medium containing rose bengal for the culturing of mycelial-agar disks after exposure to fungicides in the soil. In connection with the tests in this laboratory using the soil column method for evaluating fungicides, it was noted that the growth from mycelial-agar disks was sometimes suppressed after being subjected to a drench with certain fungicides and plated out on PDA. Thus, it might be impossible with a rose bengal medium to determine the source of the fungistasis.

Soil bacteria that contaminated the agar disks were inhibited for approximately 36 hours on the PDASV medium. This period, however, was adequate to allow viable test fungi to grow out of the disks without competition from bacterial colonies. There was seldom any contamination from air-borne bacteria in the hundreds of PDASV plates used in the soil column studies.

Literature Cited

1. BUTLER, E. E., and R. B. HINE. 1958. Use of novobiocin for isolation of fungi from soil. Soil Sci. 85: 250-254.
2. CETAS, R. C., and RODNEY WHIDDEN. 1960. Evaluation of soil fungicides against Fusarium solani isolated from feeder roots of citrus trees. Plant Disease Reptr. 44: 465-469.
3. MARTIN, J. P. 1950. Use of acid, rose bengal, and streptomycin in the plate method for estimating soil fungi. Soil Sci. 69: 215-232.
4. MEHTA, P. P., DAVID GOTTLIEB, and DWIGHT POWELL. 1959. Vancomycin, a potential agent for plant disease prevention. Phytopathology 49: 177-183.
5. NEWHALL, A. G. 1958. An improved method of screening potential soil fungicides against Fusarium oxysporum f. cubense. Plant Disease Reptr. 42: 677-679.
6. SCHATZ, A., E. BUGIE, and S. A. WAKSMAN. 1944. Streptomycin, a substance exhibiting antibiotic activity against gram-positive and gram-negative bacteria. Proc. Soc. Exptl. Biol. Med. 55: 66-69.
7. WAKSMAN, S. A. 1922. A method for counting the number of fungi in the soil. J. Bact. 7: 339-341.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF ILLINOIS,
URBANA, ILLINOIS

TRUNK CANKER OF MACADAMIA IN HAWAII CAUSED BY PHYTOPHTHORA CINNAMOMI RANDS[1]

Richard B. Hine[2]

The "smooth-shell" macadamia nut, Macadamia integrifolia, is grown as a commercial crop only in the Hawaiian Islands. Approximately 3500 acres are under cultivation with a continuing increase in acreage. Trunk and branch cankers similar to a bark disorder described from Hawaii (1) have been shown to be caused by Phytophthora cinnamomi Rands. This disease was first reported by Zentmyer from two diseased trees in California (2). In Hawaii, diseased seedling and grafted trees have been identified in several different orchards on the islands of Oahu and Hawaii. Varieties from which the fungus has been isolated include Wailua, Keauhou, and 236. A survey has shown that approximately 50 trees are diseased.

Advanced symptoms include deep furrowed trunk cankers and irregular areas of dead bark extending from the soil line to several feet high in mature trees (Fig. 1). The cankers may extend into branches. Trees are chlorotic and reduced in size compared with adjacent healthy trees of the same variety. Healthy suckers commonly sprout from the rootstock of trees in which the portion of the tree above the graft union is dead or dying.

FIGURE 1. Natural infection of Macadamia integrifolia (variety Wailua) by Phytophthora cinnamomi Rands.

Pathogenicity was proved by inoculating healthy M. integrifolia seedling trees with the fungus. One month after inoculation, small cankers were produced from which the fungus was reisolated. Only trees that were wounded at the time of inoculation developed cankers. Unwounded trees growing in artificially infested soil have not become diseased.

This newly described disease from Hawaii is potentially serious. However, damage to commercial orchards does not appear to be widespread, although a few trees have been killed by girdling action of large cankers around the trunk.

Comparative cultural studies between pineapple isolates, where the fungus causes a serious root and heart rot, and the macadamia isolate have shown minor morphological as well as temperature response differences.

The fungus was kindly examined by G. A. Zentmyer, Department of Plant Pathology, University of California, Riverside and E. J. Anderson of the Pineapple Research Institute, Honolulu, Hawaii.

Literature Cited

1. HAMILTON, R. A., and O. V. HOLTZMANN. 1958. Macadamia bark disorder discovered. Hawaii Farm Sci. 7: 3-4, July.
2. ZENTMYER, GEORGE A. 1960. Phytophthora canker of macadamia trees in California. Plant Disease Reptr. 44: 819.

HAWAII AGRICULTURAL EXPERIMENT STATION, HONOLULU, HAWAII

[1]Published with the approval of the Director of the Hawaii Agricultural Experiment Station as Miscellaneous Paper No. 143.
[2]Assistant Plant Pathologist, University of Hawaii, Honolulu, Hawaii.

TURFGRASS DISEASES IN SOUTHERN CALIFORNIA

R. M. Endo[1]

Summary

Of fungi isolated from turfgrasses in southern California and tested for pathogenicity, Helminthosporium sorokinianum is most common and destructive. Affected plants may show little leaf-spotting, but basal portions may be severely infected and rotted. H. vagans causes similar injury to Poa pratensis. Foot- and root-rot diseases caused by Olpidium brassicae, species of Fusarium, Curvularia, Pythium and Rhizoctonia also occur, but further studies are needed to define their specific roles and importance. Species of Rhizoctonia, mainly Pellicularia filamentosa and P. praticola, also infect the foliage and stems of turfgrasses usually during warm, humid weather and may cause considerable damage. H. cynodontis appears to cause little injury except to weakened plants. Sclerotinia homeocarpa, Pythium aphanidermatum, and Puccinia spp. also are prevalent and may cause substantial damage under favorable conditions. The endophytic vesicular-arbuscular mycorrhiza of Endogone (Rhizophagus), occurs commonly in roots of overwatered, heavily-matted turfgrass. The prevalent fairy-ring-forming fungi in Los Angeles County are Lepiota molybdites and Psalliota arvensis.

INTRODUCTION

In California little information is available concerning turfgrass diseases. Because numerous grass species are grown and are everywhere affected with disease, research was initiated to determine their cause, prevalence, and importance. This paper reports the fungi (and factors) associated with the deterioration, poor growth, and death of turfgrass in southern California during 1959 and 1960.

MATERIALS AND METHODS

Diseased turf was obtained from golf and bowling greens, home lawns, athletic fields, park lawns, and experimental greens. Microscopic examination was made of cleared and stained sections taken from margins of diseased tissues. These were prepared by heating in a 1:1 phenol and chloral hydrate mixture using saturated solutions of each, and containing either cotton blue or trypan blue. Isolations were made either by washing tissues in running water, or by surface sterilizing them with 1.0% sodium hypochlorite for 1 minute, followed by direct plating on water agar containing aster straw cold-sterilized with propylene oxide.

To determine if pathogens were seed-borne, seeds were soaked about 1 minute in a 1.0% sodium hypochlorite solution, air-dried on paper towels, and sown on sterilized quartz sand in Petri plates. They were watered with sterile distilled water, kept at room temperature, and examined frequently for pathogenic organisms.

Single spores or hyphal tips were isolated of fungi tested for pathogenicity and these were grown on potato-dextrose agar, autoclaved bentgrass seeds, or potato-dextrose broth. Pathogenicity was determined by two methods, using seed previously determined to be free of pathogens. In the first, seeds were sown thinly on sterile quartz sand in sterile Petri plates, and watered with sterile tap water. Seeds or 4-to-7 day old seedlings were inoculated with a spore suspension, with washed plugs of mycelium, or with a fungus-infested segment of potato-dextrose agar. Replicated plates were incubated at room temperatures or at 50°, 60°, 70°, and 80° F at a light intensity of approximately 1000 ft-c. This method, hereafter referred to as the Petri plate technique, permits the detection of weak as well as aggressive pathogens, since conditions were continuously favorable for infection and disease development. In the second method, seedlings were grown in a steam-sterilized UC sand-peat soil mix (1) in clay pots. They were inoculated with a spore suspension, or with small plugs of washed mycelium, held in a moist chamber for 48 hours, and transferred to a greenhouse bench. This method, hereafter referred to as the pot technique, was used to detect pathogens capable of attacking vigorous, healthy plants.

[1]The writer is indebted to Mrs. L. H. Davis for assistance in isolation, culture, and identification of fungi.

HELMINTHOSPORIUM LEAF SPOT AND FOOT ROT

Helminthosporium sorokinianum Sacc. ex Sorokin was most frequently isolated in spring, summer, and fall from roots, crowns, stems and leaves of Agrostis palustris (creeping bentgrass var. Seaside), Poa annua (annual bluegrass), Lolium multiflorum (Italian ryegrass), L. perenne (perennial ryegrass), Festuca rubra (red fescue var. Rainier), and Cynodon dactylon (Bermuda varieties, Common, and Ormond). It is particularly destructive as a foot- and root-rot on common Kentucky bluegrass. On the latter host, as well as on C. dactylon, F. rubra, L. multiflorum, L. perenne, and P. trivialis, symptoms are similar to those described by Christensen (4) on barley. On P. annua and A. palustris (var. Seaside), leafspotting is uncommon; instead leaves are chlorotic, later various shades of brown. The foot-rot stage, however, presents the typical light brown to chocolate brown discoloration that is found on other grass hosts.

Both seedlings and older plants are susceptible; pre-emergence damping-off is less common than post-emergence seedling blight. Seedling infections occur most commonly on cool season grasses planted out-of-season and sown at excessively high rates. Although all stages may occur at one time, leaf-spotting is more prevalent early and late in the year, while foot-rot is more common during warm months. Plants with advanced foot-rot are severely weakened and succumb readily to heat and water stress. The disease appears first in shaded areas, and is particularly damaging to common Kentucky bluegrass clipped shorter than 1 1/2 inches. Since H. sorokinianum is frequently associated in the foot-rot stage with other parasites such as Rhizoctonia solani and various species of Curvularia, Fusarium, and Pythium, diagnosis is sometimes difficult.

In pathogenicity trials with 10 isolates of H. sorokinianum involving 120 single spore cultures, the fungus was pathogenic to seedling and adult plants of A. palustris (var. Seaside), P. pratensis (var. Common), C. dactylon (var. Common), L. perenne and L. multiflorum. Although some single spore isolates appeared more pathogenic than others, all isolates produced typical symptoms.

MELTING-OUT OF KENTUCKY BLUEGRASS

Helminthosporium vagans Drechs., the cause of melting-out of Poa pratensis, was isolated commonly from this host but less frequently than H. sorokinianum. Both species have been isolated from the same bluegrass plants. Symptoms of melting-out on bluegrass correspond to the original description of Drechsler (5, 6), and the behavior of H. vagans on bluegrass is very similar to that described earlier for H. sorokinianum. However, H. vagans appears on the foliage earlier in spring and persists later into fall than H. sorokinianum. To date perithecia have not been observed.

In limited pathogenicity trials employing the Petri plate technique, 31 single spore cultures of two H. vagans isolates were pathogenic to P. pratensis (var. Common) but not to A. palustris seedlings.

HELMINTHOSPORIUM LEAF BLOTCH OF BERMUDA

Helminthosporium cynodontis Marig. is present through spring and summer on leaves, stems, rhizomes, and roots of common Bermuda. It causes little damage except on turf that is reduced in vigor because of low fertility and excess mat and thatch. It is commonly associated with the eriophyid mite, Aceria neocynodontis Keifer, which infests the buds, leaves, and sheaths, causing marked proliferation and shortening of internodes. The fungus usually attacks basal portions of mite-infested shoots, causing the tissues to turn brown.

H. cynodontis has been recovered from four samples of common Bermuda seed from Arizona; infection varied from 3 to 16%. In pathogenicity tests employing the Petri plate technique, 12 single spore cultures of H. cynodontis were mildly pathogenic to seedlings of C. dactylon, and nonpathogenic to seedlings of A. palustris, L. multiflorum, and P. pratensis.

BROWN PATCH

The brown patch disease caused by Rhizoctonia solani Kuehn (15) (Pellicularia filamentosa (Pat.) Rogers), and Pellicularia praticola (Pat.) Flentje (9) occurs most commonly during periods of high temperature (80° to 95° F) and may cause considerable damage. However, cool temperature strains of Rhizoctonia attacking bluegrass, first reported by Madison, et al. (13)

from central California, have also been isolated from southern California and found pathogenic on P. pratensis and A. palustris seedlings at a soil temperature of 55° F. Both the warm and cool temperature strains of Rhizoctonia cause symptoms similar to those described by Monteith (15) and Monteith and Dahl (16) and by Howard, et al. (8). These have been isolated from brown patch affected foliage of A. palustris, C. dactylon, F. rubra, L. multiflorum, L. perenne, P. annua, P. pratensis, and Zoysia japonica.

Leaf infections are readily diagnosed, but stem, crown and root infections are sometimes complicated by presence of other fungi such as species of Curvularia, Helminthosporium, and Fusarium. In addition, several strains of Rhizoctonia are commonly present, but not all are pathogenic. Of 34 hyphal-tip isolates tested, only 4 were pathogenic to A. palustris seedlings grown in Petri plates at 80° F.

DOLLAR SPOT

This disease, caused by Sclerotinia homeocarpa F. T. Bennett, commonly occurs on foliage of A. palustris (var. Seaside) and P. annua in golf and bowling greens along the coast of southern California (14). Creeping bent varieties that have yielded the fungus are Cohansey, Congressional, Old Orchard, Penna-Cross, and Seaside. Symptoms of dollar spot are very similar to those described by Bennett (2), and recognition of S. homeocarpa is facilitated by readily recognizable mycelia under the leaf sheath of affected plants. If favorable temperatures (68° to 85° F) prevail for several weeks, affected areas may coalesce and cause considerable damage to golf greens. The disease is easily suppressed by fungicides since mycelia are largely external.

When seven hyphal tip isolates of S. homeocarpa were tested on A. palustris seedlings at 50°, 60°, 70°, and 80° F, using the Petri plate technique, all were pathogenic.

FUSARIUM ROOT ROT

Various species of Fusarium have been isolated from browned leaves, stem bases, crowns, and roots of turfgrasses, predominantly F. oxysporum Schlecht. em. Snyd. & Hans., F. moniliforme Sheldon em. Snyd. & Hans., and F. roseum (Lk.) f. cerealis (Cke.) Snyd. & Hans. Infection was observed on seedlings more frequently than on adult plants. On the latter, Fusarium was frequently associated with species of Curvularia, Helminthosporium, Rhizoctonia and ectoparasitic nematodes, complicating diagnosis of the primary pathogen. F. nivale (Fr.) Snyd. & Hans., reported pathogenic on turf in central California (13), has not been isolated. F. roseum f. cerealis appears to be the most pathogenic on the basis of observed injury to turf. It has been isolated from reddish-brown to light brown areas of stem bases and crown of both young and old plants of A. palustris, P. annua, and P. pratensis.

Pathogenicity tests were conducted with 48 single spore cultures. Culmorum isolates of F. roseum f. cerealis were highly pathogenic to A. palustris seedlings using the Petri plate and pot techniques. Seedlings were killed in 3 weeks at 80° F. The fungus invaded the coleoptile, crown, and stem base. Sporodochia were formed in abundance. Acuminatum isolates of F. roseum f. cerealis were moderately pathogenic to A. palustris seedlings using the Petri plate technique; seedlings were not killed until 5 weeks after inoculation. The fungus invaded roots causing a light brown cortical rot. Chlamydospores were abundant, but micro- and macro-conidia were produced sparingly.

PYTHIUM ROOT ROT

Oospores and non-septate mycelia have been observed commonly in seminal and nodal roots of A. palustris, L. multiflorum, L. perenne, P. annua, and P. pratensis, under conditions of excess moisture, poor drainage, and excess mat and thatch. Pre- and post-emergence damping-off have been observed under cool and wet or warm and wet conditions, indicating that several species of Pythium may be involved. In numerous isolations, only those possessing filamentous sporangia have been obtained. P. torulosum Coker & Patterson and P. aphanidermatum (Edson) Fitzp. are the most common species isolated to date. Sporangia and oospores occur commonly in crown and coleoptile of seedlings, but only oospores have been observed in seedling roots. Older plants are stunted, the foliage is pale green, and roots are usually light brown and rotted.

P. aphanidermatum has been isolated from the leaves of A. palustris, especially when air temperatures are above 85° F. Two isolates of P. aphanidermatum were highly pathogenic to A. palustris seeds and seedlings employing the Petri plate and pot techniques at 85° and 95° F. Seedlings were slightly attacked at 80°, but not at 50°, 60°, and 70°.

FADING-OUT

The fading-out disease of turfgrass was first described by Howard and Davies (7) who attributed the disease to species of Curvularia, primarily C. lunata. In southern California Curvularia spp. have been isolated commonly from lower leaves, basal stem nodes, and roots of turfgrasses weakened by excess moisture, mat and thatch, hot weather, and chemical injury. They are frequently associated with H. sorokinianum, H. vagans, R. solani, Pythium spp. and with weak parasites and saprophytes such as H. dematiodeum, Alternaria tenuis, Stemphylium sp., Ascochyta sp. and Colletotrichum sp.

Pathogenicity trials were conducted with 45 single spore cultures of Curvularia lunata (Wakker) Bald, C. geniculata (Tracy & Earle) Bald, and C. trifolii (Kauff.) Bald, employing the Petri plate technique. After 3 weeks at 80° and 85° F, 35% kill of A. palustris seedlings occurred with C. lunata. At 60° and 70° germ tubes penetrated the coleoptile, but mycelial growth was limited and no symptoms were apparent. At 50° penetration was not observed. With C. geniculata and C. trifolii a few plants were killed at 80° and 85°.

FAIRY RINGS

Various species of basidiomycetes form fairy rings and occur rather sporadically on turf high in organic matter. According to Mr. G. F. Orr, Department of Botany, UCLA, the following species occur as rings in Los Angeles County: Lepiota molybdites (G. Meyer ex. Fr.) Sacc., Psalliota silvicola (Vitt) Fr., P. arvensis (Scharff.) Fr., P. campestris (L.) Fr., and L. naucina Fr. The last three species occur infrequently.

RUST

Leaves and culms of C. dactylon, L. multiflorum, L. perenne and P. pratensis are frequently affected with rust, particularly in temperate coastal areas. Close observation has been made of bluegrass variety plots at the University of California at Los Angeles. Rust pustules have been observed every month on the variety Merion, and under favorable conditions nearly every leaf blade may be infected, with as much as 5 to 85% of the blade covered with pustules. Newport, growing adjacent to Merion, is almost free from rust, while common Kentucky bluegrass shows much less rust than Merion.

OTHER FUNGI PRESENT IN GRASS PLANTS AND ROOTS

Typical mycorrhizal structures of the vesicular-arbuscular endophytic type, corresponding in all details to the genus Endogone (= Rhizophagus) described by Nicolson (17), have been frequently observed in roots of A. palustris, C. dactylon, L. multiflorum, L. perenne, P. annua, and P. palustris. Abundant infections occur in turfgrass roots growing in areas containing high amounts of organic matter, especially as thatch and mat.

Chytridiaceous fungi have also been observed occasionally in roots of various turfgrasses, especially under conditions of excess moisture. On some occasions cortical cells have been filled with stellate cysts of Olpidium brassicae (Wor.) Darr, and at other times with compact groups of spores that are suggestive of Ligneria.

Fungi of many genera have been observed and recovered from various turfgrasses. Genera that may be pathogenic or weakly pathogenic are Colletotrichum, Phoma, Chaetophoma, Ascochyta, Pseudoplea, Stagonospora, and Myrothecium. Saprophytic genera are Alternaria, Stemphylium, Epicoccum, Fusidium, Cladosporium, Aspergillus, Penicillium, Oedocephalum, Rhizopus, and Pullularia.

DISCUSSION

The results report a portion of the findings obtained in a program designed to study causes of poor turfgrass growth in southern California. A considerable number of fungi were isolated from leaves, stems, and roots of turfgrasses and found pathogenic. The diseases caused by Helminthosporium sorokinianum and Rhizoctonia species recur yearly and are the most serious diseases involved in stand depletion of adult plants, particularly in summer. H. sorokinianum is very destructive in the foot- and root-rot stages, as Klomparens (10) has shown in Michigan and Weihing (20) in Nebraska. H. vagans is equally destructive in the foot- and root-rot stage, but is confined to Poa pratensis and occurs less frequently than H. sorokinianum on this host.

Other fungi, such as Olpidium brassicae, species of Curvularia, Fusarium, Pythium and Rhizoctonia, also cause a rot of basal portions of turfgrasses and have been found to cause

occasional severe stand depletion. Work is in progress to determine the exact role and importance of this latter group of fungi, and various factors that determine disease occurrence and severity. Fungi causing foot- and root-rots comprise the most destructive group of pathogens in southern California. Incidence of foot- and root-rots, as well as other turfgrass diseases and troubles, is highest on cool-season grasses during hot weather. This may be related to poor root development of cool season grasses at soil temperatures above 85° F (3, 18, 19). Such plants appear to possess little tolerance to adverse environmental factors such as lack of aeration caused by overwatering or soil compaction, drought, heat stress, and to damage caused by foot- and root-rotting fungi.

Of particular interest was the common occurrence in roots of numerous turfgrasses of the endophytic vesicular-arbuscular mycorrhiza, Endogone (Rhizophagus).

Literature Cited

1. BAKER, K. F., ed. 1957. The UC system for producing healthy container-grown plants. University of California Agr. Exp. Sta. Service Manual 23, 332 pp.
2. BENNETT, F. T. 1937. Dollar spot disease of turf and its causal organism, Sclerotinia homeocarpa n. sp. Ann. Appl. Biol. 24: 236-257.
3. BROWN, E. M. 1939. Some effects of temperature on the growth and chemical composition of certain pasture grasses. Missouri Agr. Exp. Sta. Research Bull. 299. 76 pp.
4. CHRISTENSEN, J. J. 1922. Studies on the parasitism of Helminthosporium sativum. Minnesota Agr. Exp. Sta. Tech. Bull. 14. 42 pp.
5. DRECHSLER, C. 1923. Some graminicolous species of Helminthosporium. I. J. Agr. Research 24: 641-773.
6. DRECHSLER, C. 1930. Leaf spot and foot rot of Kentucky bluegrass caused by Helminthosporium vagans. J. Agr. Research 40: 447-556.
7. HOWARD, F. L., and M. E. DAVIES. 1953. Curvularia "fading-out" of turf grasses. (Abst.) Phytopathology 43: 109.
8. HOWARD, F. L., J. B. ROWELL, and H. L. KEIL. 1951. Fungus diseases of turfgrass. University of Rhode Island. Bull. 308. 56 pp.
9. KERR, A. 1956. Factors influencing the development of brown patch in lawns of Sagina procumbens L. Austral. J. of Biolog. Sciences 9: 332-338.
10. KLOMPARENS, W. 1953. A study of Helminthosporium sativum Pam., King & Bakke as an unreported parasite of Agrostis palustris Huds. Ph.D. thesis. Michigan State University. 77 pp.
11. LETEY, J. 1961. Aeration, compaction, and drainage. California Turfgrass Culture 11: 17-21. [not cited in text]
12. LUNT, O. R. 1956. A method for minimizing compaction in putting greens. Southern California Turfgrass Culture 6: 17-20. [not cited in text]
13. MADISON, J. H., L. J. PATTERSON, and T. H. HODGES. 1960. Pink snowmold on bentgrass as affected by irrigation and fertilizers. Agron. J. 52: 591-592.
14. MILLER, P. A. 1952. Dollar spot control trials. Southern California Turfgrass Culture 2: 4.
15. MONTEITH, J. 1926. The brown-patch disease of turf: its nature and control. Bull. U.S. Golf Assoc., Green Section 6: 127-142.
16. MONTEITH, J., Jr., and A. S. DAHL. 1932. Turf diseases and their control. Bull. U.S. Golf Assoc., Green Section 12: 85-187.
17. NICOLSON, T. H. 1959. Mycorrhiza in the Gramineae. I. Vesicular-arbuscular Endophytes; with special reference to external phase. Trans. Brit. Mycol. Soc. 42: 421-438.
18. STUCKEY, H. 1942. Influence of soil temperature on the development of colonial bentgrass. Plant Phys. 17: 116-122.
19. SULLIVAN, J. T., and V. G. SPRAGUE. 1949. The effect of temperature on the growth and composition of the stubble and roots of perennial ryegrass. Plant Phys. 24: 706-719.
20. WEIHING, JOHN L., STANLEY G. JENSEN, and RICHARD I. HAMILTON. 1957. Helminthosporium sativum, a destructive pathogen of bluegrass. Phytopathology 47: 744-746.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA
CITRUS EXPERIMENT STATION, RIVERSIDE, CALIFORNIA

THE RELATION OF PLANT FERTILITY TO BACTERIAL SPOT OF PEPPER[1]

W. W. Townsley and D. F. Crossan[2]

Summary

Greenhouse studies with potted pepper plants indicated that a high fertility level was effective in delaying the abscission of leaves severely infected with Xanthomonas vesicatoria but was not effective in reducing the incidence of leaf spotting.

INTRODUCTION

Bacterial leaf spot of pepper, caused by Xanthomonas vesicatoria (Doidge) Dowson, has continued to be a problem in Delaware during recent years. Its incidence and severity is associated with prolonged damp periods accompanied by wind-driven rain.

Infection of the pepper fruit results in a reduction of quality. Severe infection of the foliage causes yellowing of the leaves followed by defoliation, thus exposing the fruit to possible sunscald. In most cases sunscald results in a greater reduction of marketable yield than fruit infection caused by X. vesicatoria. An economic method for the reduction of the damage by bacterial spot is of the utmost importance for continued pepper production.

In 1960 Taylor (3), and Taylor and Dobson (4), reported that high fertility levels were effective in reducing the incidence of pepper bacterial leaf spot. Nayudu and Walker (2) reported similar results with bacterial spot of tomato, caused by the same organism. Crossan, et al. (1) reported that under field conditions high fertility levels were effective in reducing the defoliation of pepper due to infection caused by X. vesicatoria.

MATERIALS AND METHODS

During the period October 1960 to April 1961, experiments were conducted in the greenhouse on potted pepper plants to determine the effect of fertility levels on the incidence of bacterial leaf spot and the abscission of infected leaves. Plants of the California Wonder variety were grown in glazed crocks containing screened field soil. The crocks had a 1-inch drainage hole at the bottom. A given series of test plants were watered daily with one of three concentrations of a modified Hoagland's solution (Table 1) from transplanting to time of first flowering.

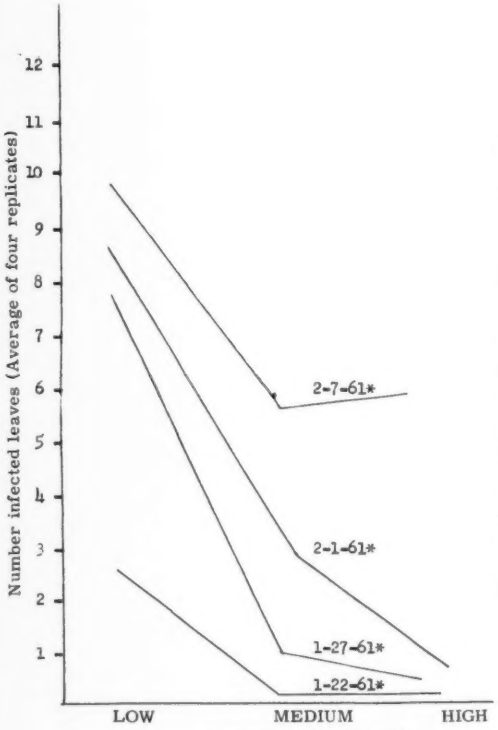
Table 1. Chemicals used in the nutrient solutions to provide pepper plants with low, medium, and high nutritional levels.

Chemicals	Amounts per liter for nutrient levels		
	Low	Medium	High
$Mg(NO_3)_2$	0.0427g	0.2564g	0.5128g
$CaSO_4$	0.0233g	0.1400g	0.2800g
NH_4NO_3	0.0400g	0.2400g	0.4800g
KN_2PO_4	0.0341g	0.2043g	0.4083g
$Ca(NO_3)_2$	0.1575g	0.9448g	1.8896g
K_2SO_4	0.0871g	0.5226g	1.0452g
$MgSO_4$	0.0041g	0.0246g	0.0492g
Minor elements[a]	3.0 ml	3.0 ml	3.0 ml
Fe(iron supplied by ferric disodium ethylene diamine tetra-acetate (50g/l))	3.0 ml	3.0 ml	3.0 ml

[a] Composition of minor element stock solution (g/l): H_3BO_2, 2.86; $MnCl_2$, 1.81; $ZnSO_4$, 0.22; $CuSO_4$, 0.08; H_2NoO_4, 0.09.

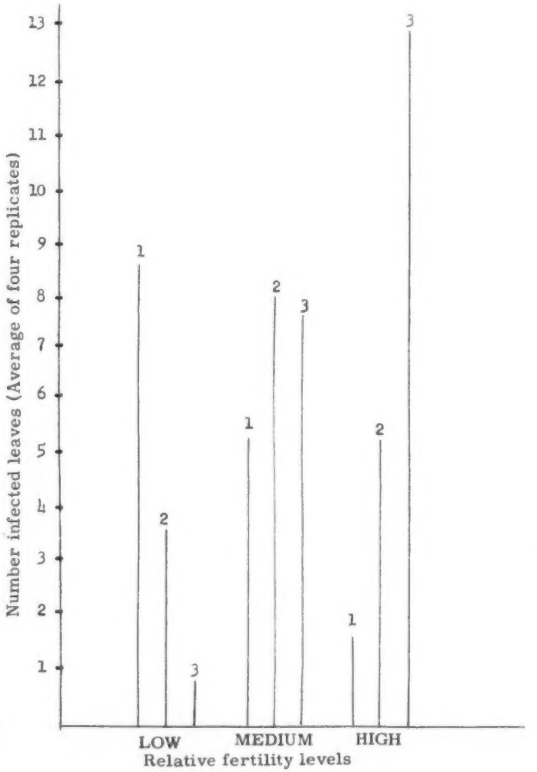
[1] Published as Miscellaneous Paper No. 403 with the approval of the Director of the Delaware Agricultural Experiment Station. Contribution No. 141 of the Department of Plant Pathology.

[2] Graduate Assistant and Associate Professor, respectively.



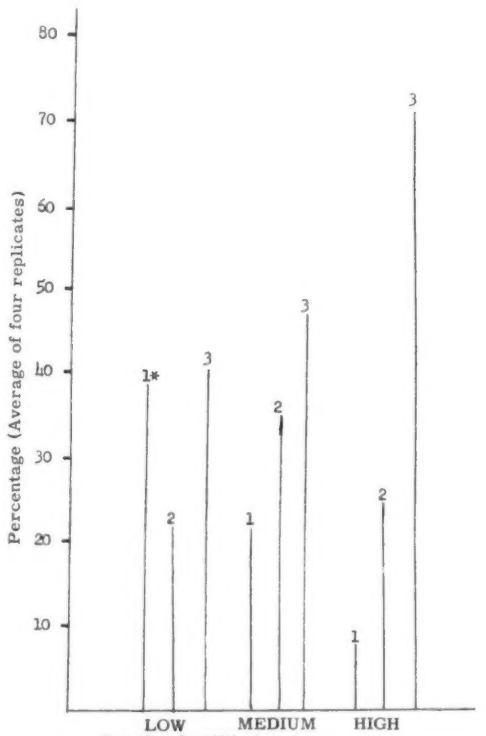
*Dates indicate scoring periods. Plants inoculated 1-5-61; first leaf fall 1-18-61.

FIGURE 1. Effect of fertility levels on the time of abscission of pepper leaves infected with X. vesicatoria in greenhouse tests. Newark, Delaware, 1960.



*Infection categories (lesions per leaf): 1, 0-49; 2, 50-99; 3, 100 or more.

FIGURE 2. Effect of fertility levels on number of pepper leaves infected with X. vesicatoria remaining on plant 32 days after inoculation in greenhouse tests. Newark, Delaware, 1961.



Infection categories (lesions per leaf); 1, 0-49; 2, 50-99; 3, 100 or more.

FIGURE 3. Effect of fertility levels on percentage of total infected leaves in each infection category of plants grown at low, medium, and high fertility levels in greenhouse tests. Newark, Delaware, 1961.

Each treatment was replicated four times. At the time of first flowering, the upper and lower leaf surfaces were sprayed with a suspension of the bacteria that had a reading of 50% light transmission on a Fisher electrophotometer fitted with a number 425 blue filter. Spraying of the suspension was continued until the lower surface of the leaves was spotted with water-soaked areas. A paint spray gun, attached to a portable air compressor, was used to apply the suspension.

RESULTS

Blister-type symptoms appeared on lower leaf surfaces approximately 10 days after inoculation. In low fertility level plants such lesions enlarged rapidly and by 1 week after the appearance of the initial symptoms they were circular, necrotic spots. In contrast, the blister-type lesion on high fertility level plants remained as small, blistered areas for 3 to 4 weeks before exhibiting a necrotic area visible on both the upper and lower leaf surfaces.

Prior to the tests, the infected leaves were divided into three categories based on the severity of infection: 1, 0-49 lesions per leaf; 2, 50-99; 3, 100 or more. As the first and subsequent leaves abscised, they were ranked by infection category and fertility level (Figs. 2, 3). The effect of fertility levels on the time of abscission of infected leaves is shown in Figure 1.

When leaf abscission finally increased on plants grown at high fertility levels, the test was terminated. A count of the infected leaves remaining on the plants at the end of the experiment (Fig. 2) showed that the largest number of infected leaves on the plants grown at low levels of nutrition were of the slightly infected category (0-49 lesions per leaf) and the smallest number of leaves were of the severely infected category (100 or more lesions per leaf). The reverse of this was true on plants grown at high nutritional levels. The largest number of infected leaves remaining on the plants grown at high nutritional levels were in the severely infected category and the smallest number were in the slightly infected category. The percentage of the total number of infected leaves (those that fell and those on the plants at the end of the experiment) in each infection category is shown in Figure 3. There was no predominance of infected leaves in any infection category on plants grown at low nutritional levels. On plants grown at high nutritional levels, there was a marked predominance of severely infected leaves and almost no leaves in the slightly infected category.

DISCUSSION

The greenhouse tests conducted during 1960-1961 indicated that high fertility levels were effective in retarding necrotic symptom expression and delaying the abscission of pepper leaves infected with X. vesicatoria but were not effective in reducing the severity of leaf spotting. Plants grown at high nutritional levels had a higher incidence of leaf spot than plants grown at low nutritional levels but retained infected leaves for a longer period of time.

Literature Cited

1. CROSSAN, D. F., D. J. FIELDHOUSE, P. P. BURBUTIS, W. W. TOWNSLEY, Jr. and ROBERT VANDENBURGH. 1961. Control of pepper bacterial spot by fertilizer and by foliar sprays. Plant Disease Reptr. 45: 120-123.
2. NAYUDU, M. V., and J. C. WALKER. 1960. Bacterial spot of tomato as influenced by temperature and by age and nutrition of the host. Phytopathology 50: 360-364.
3. TAYLOR, J. 1960. Studies on the relation of fertilization to development of bacterial and Cercospora leaf spot in pepper. (Abst.) Phytopathology 50: 577.
4. TAYLOR, JACK and J. W. DOBSON, Jr. 1960. The relation of fertilization to development of bacterial spot on pepper. Plant Disease Reptr. 44: 172-175.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF DELAWARE,
NEWARK, DELAWARE

APHIS GOSSYPII, A VECTOR OF CITRUS VEIN-ENATION VIRUS

E. F. Laird, Jr. and L. G. Weathers[1]

INTRODUCTION

Citrus vein-enation virus has been shown to be transmitted by the brown citrus aphid, Toxoptera citricidus (Kirk.), in South Africa (1) and by the green peach aphid, Myzus persicae (Sulz.), in California (2). Wallace and Drake (2) were unable to demonstrate transmission of the virus from Mexican lime or sweet orange to Mexican lime seedlings with the melon aphid, Aphis gossypii Glover. Following the report of the severe synergistic interaction between the viruses of yellow vein and vein enation when they were combined in citrus plants (3), the writers deemed it important to determine if these two viruses might be transmitted simultaneously by insect vectors. This paper reports the results of preliminary transmission studies with these viruses by M. persicae and A. gossypii.

Mexican lime plants, Citrus aurantifolia, singly-infected with vein-enation virus and Rough lemon plants, C. jambhiri, and Mandarin plants, C. reticulata, doubly-infected with vein-enation virus and yellow-vein served as source plants. All transfers were made to Mexican lime seedlings. Aphis gossypii was reared on watermelon in the laboratory and transferred on watermelon leaves directly to the virus source plant for an acquisition feeding time of 3 to 7 days. M. persicae was reared on pepper plants and handled similarly to A. gossypii. From the virus source plant the aphids were placed on healthy test plants and confined within cotton, sleeve-shaped cages. The number of aphids used for each test varied from 10 to 500, with an average of about 110. After 3 to 7 days of confinement on the test plant, the aphids were killed with nicotine fumigant. Test plants were held in a greenhouse maintained at about 78° F to await symptom development.

Yellow-vein virus was not transmitted from plants doubly-infected with yellow-vein and vein-enation viruses by either of the insect species used in these tests. Two of 34 Mexican lime seedlings infested with M. persicae became infected with vein-enation virus. In both instances the vein-enation virus was acquired from plants infected with both yellow-vein and vein-enation viruses. These results confirm the work of Wallace and Drake (2) that M. persicae is a vector of vein-enation virus.

Four of 11 Mexican lime seedlings infested with A. gossypii developed symptoms of vein enation. In two instances the vein-enation virus was transmitted from plants doubly-infected with yellow-vein and vein-enation viruses. Vein enation symptoms were seen within 18 to 33 days following aphid transfers to the test plants.

McLean (1) demonstrated that vein-enation virus in citrus in South Africa is transmitted by T. citricidus, the principal vector of tristeza virus in South Africa. A similar situation occurs in California where A. gossypii, the vector of tristeza virus, can also transmit the vein-enation virus. Other than being a vector of vein-enation virus, little is now known of the relationship of A. gossypii to the virus. Aphis gossypii is widespread and occurs in large populations in southern California citrus groves. The knowledge that A. gossypii, in addition to M. persicae, is a vector of vein-enation virus and that both species occur in large populations perhaps may explain the high incidence of vein-enation virus observed in southern California groves in recent years.

Wallace and Drake (2) frequently recovered the citrus vein-enation and tristeza viruses from the same tree. Since both viruses have the same vector, A. gossypii, it would be of interest to determine if this aphid would transmit the viruses simultaneously or if the presence of one virus had an effect on the vector's ability to transmit the other. Also, through insect transmission, the effect, if any, of one virus on the other within the host might be determined.

Literature Cited

1. McLEAN, A. P. D. 1954. Citrus vein-enation virus. S. African J. Sci. 50: 147-151.
2. WALLACE, J. M., and R. J. DRAKE. 1959. Citrus vein enation. In Citrus Virus Diseases, edited by J. M. Wallace, University of California, Div. of Agr. Sciences. pp. 163-165.
3. WEATHERS, L. G. 1960. Yellow-vein disease of citrus and studies of interactions between yellow-vein and other viruses of citrus. Virology 11: 753-764.

UNIVERSITY OF CALIFORNIA CITRUS EXPERIMENT STATION, RIVERSIDE, CALIFORNIA

[1]Laboratory Technician IV and Associate Plant Pathologist, Citrus Research Center and Agricultural Experiment Station, Riverside, California. The authors wish to express their thanks to M. K. Harjung for valuable technical assistance.

SOURCES OF RESISTANCE TO THE COWPEA YELLOW MOSAIC VIRUS[1]

Darrell G. Wells and Raphael Deba[2]

Summary

One hundred sixteen cowpea varieties introduced from America and 342 indigenous pure-lines were tested against the cowpea yellow mosaic virus. Six varieties and 16 indigenous pure-lines were resistant.

INTRODUCTION

Cowpea yellow mosaic is the most important disease affecting cowpeas, Vigna unguiculata, in Western Nigeria, sometimes reducing seed yields to nothing in entire farm seedlings. In an effort to develop adapted varieties resistant to the cowpea yellow mosaic virus all available pure-lines of cowpeas were inoculated.

Chant (1) characterized the cowpea yellow mosaic virus and distinguished between it and the other viruses on cowpea described in the literature. Chant (2) also determined the influence of cowpea yellow mosaic on growth rate and yield of cowpeas.

MATERIALS AND METHODS

The cowpeas tested consisted of the world collection of varieties[3] of the United States Department of Agriculture of which viable seed of 116 remained to us from an original group of 138 varieties. Also tested were the indigenous collections made in markets and on farms in Nigeria and the Southern Cameroons and available from the breeding nursery as pure-lines. There were 89 numbered indigenous collection groups distinguished from one another in seed color or in area of origin and consisting of 342 pure-lines.

All entries were seeded in seven hills 2 inches apart, one or two seeds per hill. Two of the hills served as checks. Five hills were inoculated in the unifoliate leaf stage by rubbing the leaves with inoculum mixed with sand in which the seedlings grew until the leaf surface was visibly scratched or broken. Inoculum was supplied by Mr. Gary Robertson, Federal Pathologist, Moor Plantation, in the form of infected cowpea seedlings showing the characteristic systemic symptoms of vein clearing and interveinal chlorosis. Inoculum was prepared by maceration of an infected seedling, filtering of the expressed infective sap through cotton cloth, and dilution with tap water to about 1 in 800. The seedlings were pulled and classified about 4 weeks later. Reaction classes (Fig. 1)[4] were the following:

1. No symptoms.
2. Pale mottling and slight crinkling of some leaves, slight dwarfing or reduction in stem diameter.
3. Moderate dwarfing and mottling, or pronounced mottling alone.
4. Severe mottling and sometimes severe crinkling of leaves and moderate dwarfing, severe dwarfing but leaves almost normal in color although reduced in size.
5. Severe chlorosis, crinkling and distortion of leaves, severe dwarfing, or seedlings dead.

Disease indices were calculated by multiplying the number of seedlings in a class times the value of the class, summing all such products for a variety and dividing by the number of inoculated seedlings. The class values were 0, 25, 50, 75, and 100 for classes 1 to 5 respectively.

In the earlier tests, Paraguay 6 was used as a resistant check because it had exhibited field tolerance or resistance. However, this quality was not demonstrated when seedlings of Paraguay 6 were inoculated in the two-leaf stage so the readings made on the Paraguay 6 checks in the test reported in Table 1 are omitted. The susceptible check was an indigenous mixed market collection, V105, of which about 25,000 seedlings had been screened earlier for resistance without success. In the later tests, Arlington was the resistant check and New Era the susceptible check.

---

[1]Contribution from the Ministry of Agriculture, Western Region, Ibadan, Nigeria, and the International Development Services, Inc., New York.

[2]Professor of Agronomy on leave of absence from Mississippi State University, and Agricultural Superintendent, Nigerian Western Region Ministry of Agriculture, respectively.

[3]Supplied by Dr. Edwin James, Experiment, Georgia.

[4]The authors are indebted to Mr. J. O. Bukota, Federal Pathologist, Moor Plantation, Ibadan, for developing the film and printing the pictures.

FIGURE 1. Examples of plants in classes 1 through 5 for reaction to the cowpea yellow mosaic virus.

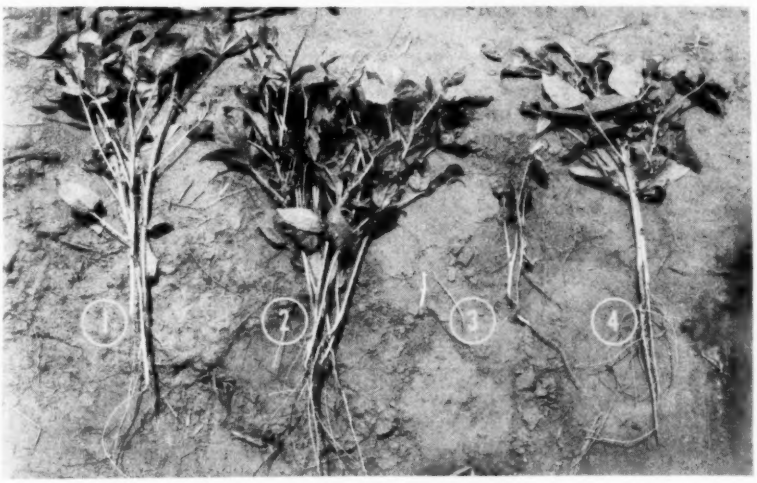


FIGURE 2. Reactions to the cowpea yellow mosaic virus of two indigenous purelines of the same seed type: 1 and 2 -- check and inoculated plants of a moderately resistant line, 3 and 4 -- inoculated and check plants of a susceptible line.

Apparent reactions were indicated as follows:

Classes	Ranges of mean indices	
	In the first tests	In subsequent tests
R-resistant	0-29	0-20
MR-moderately resistant	30-55	21-40
MS-moderately susceptible	56-70	41-60
S-suseptible	71-100	61-100

RESULTS

Reactions to the cowpea yellow mosaic virus of a susceptible check variety and the resistant varieties among 116 entries[5] are shown in Table 1. The susceptible check variety reacted as anticipated. Paraguay 6, Victor, Clay Brown 1330, Clay Buff, and Early Red K736 were classed as moderately susceptible or susceptible because of being MS or S in one of the three replicates and therefore were not tested further. Of 13 varieties that were moderately resistant,

[5]A complete list of the varieties tested and their reactions can be obtained from the senior author.

remnant seed was available of nine. These were tested again against the virus with the results shown in Table 2. Resistance was confirmed for Arlington, Jackson Alabama, Victor K798, Groit, Brabham Victor, and Brabham K892.

Fifty-nine of the 342 indigenous pure-lines were considered resistant (Fig. 2). Remnant seed was available of 56 of these and they were tested once more for confirmation of their favorable reactions. Results of both tests on the surviving 16 lines are given in Table 3 together with origin of the original heterogeneous seed lots and the seed type of the pure-lines.

Table 1. Reactions to the cowpea yellow mosaic virus of the varieties in the world collection of cowpeas that were resistant.

Variety	Indices				Apparent reaction
	Rep. I		Rep. II		
	inoculated	check	inoculated	check	
V105, check	100	15	100	0	S
Red Ripper	31	0	50	0	MR
Arlington	40	0	46	0	MR
Jackson Alabama	33	0	25	5	MR

Variety	Rep. I		Rep. II		Rep. III		Apparent reaction
	inoculated	check	inoculated	check	inoculated	check	
V105, check	100	0	100	38	100	8	S
Clay 1	45	0	53	33	44	25	MR
Clay Brown Okla. 992	34	13	46	8	28	25	MR
Early Red	38	17	33	25	25	25	MR
Red Ripper K711	38	8	34	20	38	21	MR
Iron K329	--	--	50	--	--	--	MR
Victor K798	23	0	33	25	25	15	MR
Whippoorwill	25	25	36	20	38	0	MR
Groit	25	8	41	12	31	25	MR
Brabham Victor	43	6	--	--	32	19	MR
Brabham K892	25	0	42	17	30	10	MR

Table 2. Confirmatory test of the varieties in the world collection of cowpeas that were resistant to the cowpea yellow mosaic virus in the first test.

Entries	Indices by replicates			$\bar{x}$	Indicated reaction
	I	II	III		
New Era, susc. check	79	80	75	78	S
Arlington	29	25	25	26	R
Jackson Alabama	25	25	19	23	R
Early Red	100	100	50	83	S
Red Ripper K711	88	--	81	85	S
Victor K798	13	25	45	28	MR
Whippoorwill	--	--	75	75	S
Groit	--	8	25	17	R
Brabham Victor	40	33	25	33	MR
Brabham K892	25	38	10	24	R

Table 3. Indices in consecutive tests of reaction to the cowpea yellow mosaic virus of a group of indigenous pure-lines and their origins and seed colors.

Variety or selection	Original seed source	Seed type	Test 1	Test 2	$\bar{x}$	Apparent reaction
Arlington, res. check	USA	White, browneye, smooth, small	35	36	36	MR
New Era, susc. check	USA	Blue mottle on brownish black, medium size, smooth	75	79	77	S
V32-4	Obu Nkete		--	36	36	MR
V45-1	Western Region	Diffuse blue speckle on tan, small, smooth crowder	21	48	35	MR
V46-2	Ibadan District market	Tan, small, smooth crowder	33	17	25	MR
V136-2	Northern Region markets	Diffuse black speckle on white, blackeye, medium size, wrinkled crowder	27	25	26	MR
V141-4	Kaduna market	Diffuse brown on tan, small, smooth crowder	25	38	32	MR
-5	do.		29	38	34	MR
V147-3	do.	Diffuse brown on tan, small, smooth crowder	31	47	39	MR
V153-1	Sapele, Southern Cameroons	Tan to brown, medium size, smooth crowder	42	38	40	MR
V154-8	do.	Red, medium size, smooth crowder	30	46	38	MR
V155-1	do.	Diffuse brown on tan, medium size, smooth crowder	33	43	38	MR
-2		do.	33	25	29	MR
-3		do.	28	29	29	MR
-4		do.	29	20	25	MR
-6		do.	34	25	30	MR
-9		do.	25	37	31	MR
V165-5	Ibadan District market	Blackeye, large wrinkled crowder	25	--	25	MR

Literature Cited

1. CHANT, S. R. 1959. Viruses of cowpea, Vigna unguiculata L. (Walp.), in Nigeria. Ann. Appl. Biol. 47(3): 565-572.
2. CHANT, S. R. 1960. The effect of infection with tobacco-mosaic and cowpea yellow-mosaic viruses on the growth rate and yield of cowpea in Nigeria. Empire J. of Exp. Agr. 28(110).

MISSISSIPPI STATE UNIVERSITY, STATE COLLEGE, MISSISSIPPI AND NIGERIAN WESTERN REGION MINISTRY OF AGRICULTURE, IBADAN, NIGERIA

OCCURRENCE OF VIRUSES CAUSING RASPBERRY MOSAIC IN SOME COMMERCIAL STOCKS OF RED RASPBERRY IN EASTERN UNITED STATES

Richard H. Converse[1]

Summary

The occurrence of viruses causing raspberry mosaic varied from 17 to 100% of the stocks of individual varieties when 85 samples of superior stocks of 7 red raspberry varieties were obtained from several places in eastern United States. Approximately one-third of all stocks from these 7 varieties were carrying raspberry mosaic viruses in a symptomless condition, as determined by indexing on black raspberry seedlings. A list is given of the Rubus varieties so far assembled by the United States Department of Agriculture that are free from the viruses detectable by the currently used indexing procedures.

INTRODUCTION

Investigations were initiated in 1957 by the United States Department of Agriculture designed to determine whether virus-free stocks might be found and used in certified Rubus stock programs in the United States. Stocks of leading red raspberry varieties received from growers, nurserymen, and agricultural officials from various localities in eastern United States were indexed for viruses causing raspberry mosaic during 1958 to 1960. Plants free from raspberry mosaic symptoms and free from symptoms of curl, which is not known to be latent in red raspberry, were indexed by attempted transmission to indicator plants. They were considered free from raspberry mosaic viruses if no symptoms appeared on the indicator plants, but they might harbor other viruses not detected by the indexing procedures used. This paper reports the results of this red raspberry indexing and lists the collection of red and black raspberry varieties that have so far been found to be free from known viruses.

MATERIALS AND METHODS

Red raspberry varieties from various locations planted in the field at Beltsville, Maryland were kept free from aphids by biweekly applications of parathion during the growing season. Plants were rated in the field for raspberry mosaic symptoms and were also indexed by procedures patterned after described techniques (1, 2). Detached young shoot tips of plants to be indexed were held in Petri dishes in the laboratory. Virus-free Amphorophora rubi (Kltb.) were placed on the shoot tips for about 24 hours. Usually about 10 aphids were then moved with a small brush to each of 3 to 5 small potted black raspberry seedlings (Rubus occidentalis). Open-pollinated seedlings of the black raspberry varieties Plum Farmer, Cumberland, and the selection Md-16 x 18 were all satisfactory and were used interchangeably. Black raspberry indicator seedlings were observed for 6 weeks for symptom development and were then discarded.

Stocks shown to be free from raspberry mosaic viruses by repeated indexing with aphids were further tested by leaf-grafting to black raspberry seedlings before being increased.

RESULTS

Five aphids per test seedling transferred from a known source gave 88% transmission of viruses of the raspberry mosaic complex. It thus seems that repeated indexing with lots of five aphids or more from suspects is a good test procedure to determine the presence of raspberry mosaic viruses. The initial tip-necrosis symptoms of raspberry mosaic usually developed in 7 to 10 days, followed by mottling and dwarfing. Vein clearing like that previously described (2) appeared in some cases after 3 to 5 weeks.

The occurrence of raspberry mosaic viruses in seven varieties of red raspberry, five or more clones of which were indexed, is listed in Table 1. While no varieties were entirely free, several individual stocks of some varieties were free from raspberry mosaic viruses. No

[1]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of agriculture, Beltsville, Maryland. The assistance of the many State Department of Agriculture and State Agricultural Experiment Station workers who cooperated in this study is gratefully acknowledged.

Latham stocks were free and only one of seven Taylor stocks was healthy. Raspberry mosaic viruses were present without symptoms in approximately one-third of the 85 stocks examined of all varieties.

Table 1. Occurrence of raspberry mosaic viruses in 7 red raspberry varieties from eastern United States, 1958-1960.

Variety	No. of States from which stocks were tested	Type of infection (number)		
		visibly infected	symptomless but carrying mosaic	mosaic-free
Durham	4	0	4	3
Indian Summer	4	0	2	4
Latham	9	26	12	0
Milton	1	0	1	5
Newburgh	5	2	4	5
September	5	0	2	8
Taylor	3	3	3	1

One or two stocks of each of 17 additional red raspberry varieties were indexed, and stocks of nine of these varieties (Antietam, Canby, Cuthbert, Herbert, Newman, Ontario, Rideau, Washington, and Willamette) were found that were free from raspberry mosaic viruses.

DISCUSSION

A wide range of symptoms was encountered on the inoculated black raspberry seedling indicator plants, including shoot-tip necrosis followed by mottling and dwarfing, rapid development of mottling without necrosis, and delayed vein clearing resembling that described by Stace-Smith (2). It is not the purpose of this paper to resolve the possible strains or component viruses found and relate them to those already described (3). The term "raspberry mosaic" is used to indicate the symptoms produced by all viruses encountered in the indexed stocks (Table 1) that were transmitted by A. rubi to black raspberry seedlings.

A search for stock of Latham, the most widely grown red raspberry in the United States, which might be free from the viruses causing raspberry mosaic, was a primary objective of the study. Latham from 38 locations in nine States, including some stocks maintained as long as 40 years by the same grower, were examined, and all clones contained viruses of the raspberry mosaic complex, although not all showed symptoms. Virus contamination of Latham probably occurred early in its distribution and may be universal in this variety.

Most of the plants obtained for testing were from outstandingly vigorous stocks known to the State officials in the areas. The prevalence of symptomless virus-infected stocks of red raspberry poses serious problems to growers and to State certification officials concerned with producing and maintaining healthy stocks. Field inspections and roguing are not adequate for certification of red raspberry stocks as being free from viruses of the raspberry mosaic complex.

Clones of red and black raspberry that are free from raspberry mosaic viruses were obtained as a result of these studies, and additional ones were obtained from the Canadian Department of Agriculture. These stocks are currently being maintained by the United States Department of Agriculture at Beltsville, Maryland. Varieties that have been propagated and distributed to various State agencies include Canby, Cuthbert, Durham, Indian Summer, Milton, Newburgh, Rideau, September, Taylor, and Willamette red raspberries; and Bristol, Cumberland, Dundee, Morrison, and New Logan black raspberries.

Investigations are being continued to increase the collection of Rubus stocks free from raspberry mosaic viruses and other known viruses. Heat treatments are being used in an attempt to obtain virus-free plants of varieties like Latham which appear to be universally affected.

Literature Cited

1. HUBER, GLENN A. 1954. Technique for indexing red raspberry plants for viruses. Plant Disease Reptr. 38: 68-69.
2. STACE-SMITH, R. 1955. Studies on Rubus virus diseases in British Columbia. I. Rubus yellow-net. Can. J. Botany 33: 269-274.
3. STACE-SMITH, R. 1956. Studies on Rubus virus diseases in British Columbia. III. Separation of components of raspberry mosaic. Can. J. Botany 34: 435-442.

PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND

SOIL-BORNE MOSAIC OF FALL-SEEDED OATS IN WESTERN WASHINGTON[1]

G. W. Bruehl and V. D. Damsteegt

Abstract

A mosaic of fall-seeded oats (Avena sativa, A. byzantina) developed in three successive seasons at Vancouver, Washington. Winter wheat, rye and barley and spring oats, wheat and barley in the same fields were unaffected. Transmission with expressed juice and carborundum was difficult from oats to oats, and unsuccessful to all other plants inoculated (several Gramineae, some members of Chenopodiaceae, Compositae, Cruciferae, Cucurbitaceae, Leguminosae, Rosaceae, Solanaceae, Tropaeolaceae, Umbelliferae). Aphids (Macrosiphum dirhodum, M. granarium, Rhopalosiphum fitchii) did not transmit and eriophyid mites were not found on the oats. Symptoms ranged from flecks to streaks to well developed eye-spot type mottlings. Mosaic developed in greenhouse-grown oats seeded in soil from the affected fields. The virus is not seed-borne. Symptoms on plants seeded in infested soil appeared 90 or more days after sowing. The Lolium virus of Western Washington produces similar symptoms on oats, but the soil-borne oat virus did not infect Lolium spp., either in the field or in transmission trials. Host range, symptoms, and field behavior suggest similarity with the soil-borne oat mosaic of southeastern United States, but little or no relationship to the soil-borne tomato black ring virus (TBRV) and its beet ringspot strain of England which can also produce systemic infections in oats.

INTRODUCTION

A mosaic producing chlorotic dashes, streaks and "eye-spot" (10) type mottles on fall-seeded cultivated oats (Avena sativa and A. byzantina) was observed in plots at the Southwestern Washington Experiment Station, Vancouver, from February to June 1959. Several winter wheats (Triticum aestivum), wheat hybrids (hybrids of common wheat and T. durum, T. dicoccum, T. dicoccioides, T. persicum, T. polonicum, T. timopheevi) and fall-seeded rye, as well as Lolium spp. growing as weeds, were free of mosaic symptoms. Approximately 4000 varieties of oats, many other Avena species, and over 1000 barleys seeded in the same field in April of 1959 likewise were free of mosaic of any kind.

FIELD OBSERVATIONS

In winter and spring of 1960 mosaic was again seen in winter oats and in fall-germinated volunteer spring oats, the commercial variety Shasta, in a second field on the same experimental farm. In this same field were over 3300 varieties of winter wheat, 1200 varieties of winter barley, a few rows of winter rye, and several hundred varieties of spring oats, wheat, and barley, as well as a heavy growth of Lolium multiflorum as a weed. For the second year mosaic occurred only in fall-seeded oats.

The mosaic was prevalent in and severe on Tech, although yellow dwarf (barley yellow dwarf virus) was rampant in this nursery. Schwarzhafer, C. I. No. 4052, S-81, and Black Winter oat were all so severely diseased with mosaic that these varieties were recorded "too poor to read" for yellow dwarf. They were more or less rosetted with many dead tillers. It is impossible to assess the separate contributions of the mosaic and yellow dwarf on these oats, but it is highly probable that the latter three exhibit extreme susceptibility to the soil-borne oat mosaic. Varieties of fall-seeded oats on which mosaic was observed are as follows:

P. I. No.'s 174520, 177769, 185658, 185780. C. I. No.'s 691 Appler, 947 Tech, 2042 Lee, 2499 Pentagon, 2505 Hairy Culberson, 3296 Winter Turf, 3370 S-82, 3378, 3520 Schwarzhafer (1671), 3976 Shasta (Volunteer), 4052, 4107 S-81, 4108 Black Winter Oat, 4212, 4468 Bancroft, 5364 New York Sel., 5382, 5601, 6572 Dubois, 6980 Ballard:Ky. 45-34, 7132, 7214, 7215, 7216, 7221, 7222, 7223, 7296, 7297, 7298, 7299, 7300, 7301, 7302, 7303.

Limited efforts to transmit the causal entity from the diseased fall oats to adjacent seedlings of spring oats in the field by means of expressed juice and carborundum failed. Likewise, no

[1]Scientific Paper No. 2155, Washington Agricultural Experiment Stations. Project 1546.

visible evidence of transmission resulted from the use of aphids (Macrosiphum dirhodum (Walker), Rhopalosiphum fitchii (Sanderson), Macrosiphum granarium (Kirby)) as possible vectors. No eriophyid mites were found on the diseased oats. No bacteria or fungi were associated with the diseased oats, so far as could be determined.

The only known mosaic of oats in the United States that affects mainly fall-seeded oats rather than spring oats, and that does not produce symptoms on the other winter cereals, is the soil-borne oat mosaic of the southeastern section of the United States (5, 10, 12). The eye-spot type lesion on several varieties was indistinguishable from the eye-spot type of soil-borne oat mosaic described by McKinney (10). The failure to transmit in limited attempts, either with aphids or carborundum and expressed juice (10, 12), also suggested affinity with the mosaic of the Southeast. The failure of this disease to appear in the many adjacent spring-seeded varieties of oats, some the same varieties as in the fall oat nursery, indicated the probable absence of an active aerial vector. With this circumstantial evidence experiments were performed to determine whether this mosaic is indeed incited by a soil-borne entity.

EXPERIMENTS TO DETERMINE THE SOIL-BORNE NATURE OF THE PATHOGENIC ENTITY

Seed of the most severly diseased varieties of oats was saved in the summer of 1960, and soil was removed in August to a depth of about 6 inches directly below diseased oats. This soil was dry and hard. It was brought to a moisture content rendering it friable, screened to remove coarse roots and crown tissues, and sacked for use when needed. It was stored in the greenhouse headhouse.

Experiment 1: On August 14, 1960 six-inch clay pots were filled with the suspect soil. Half of the pots were then steamed at 8 psi for 2 hours. When the soil had cooled, masses of the dried roots from the screening process were introduced into three of the steamed pots. All pots were then seeded with seed saved from diseased plants. As cool temperatures favor development of the southeastern soil-borne oat mosaic (10, 11, 12), the pots were incubated in growth chambers at 55° F for 2 months, then transferred to a greenhouse maintained at approximately 65° for the duration of the experiment.

Mosaic first appeared December 18, 126 days after sowing, in oats in a pot of unsteamed soil. From that time on more and more plants developed symptoms (Table 1). No symptoms appeared in the oats planted in steamed soil, not even when root masses were added.

Table 1. Approximate period from seeding until appearance of soil-borne Western Washington oat mosaic in pot experiments. Recordings of new plants or shoots developing mosaic were ended February 4, 1961.

Experiment 1 Symptoms recorded			Experiment 2 Symptoms recorded		
Date	Days after seeding	No. of new diseased plants on that day	Date	Days after seeding	No. of new diseased plants on that day
Dec. 18	125	1	Dec. 27	93	1
20	127	2	Jan. 5	101	1
26	133	1	6	102	1
31	138	1	8	104	2
Jan. 3	141	1	20	116	7
5	143	8	26	122	4
6	144	1	Feb. 4	130	5
8	146	2			
9	147	2			
20	158	10			
22	160	3			
23	161	2			
Feb. 4	172	44			

Experiment 2: Suspect soil was placed in 6-inch clay pots out-of-doors from September 24, 1960 to October 28 when half of them were brought into the greenhouse and to November 19 when the remainder were brought in. The first plant with mosaic was seen on December 27, 93 days after seeding. Subsequently, the number of plants with mosaic gradually increased (Table 1). Some plants in all pots eventually developed mosaic.

Experiment 3: Tomato black ring virus (TBRV)-infected soil is reported to lose its infectivity if it is dried for 1 week at 20° C (3). As beet ringspot virus (6, 7), a strain of TBRV, is known to infect oats, it was feared that the dry soil might lose its infectivity during storage. Fresh soil was obtained from the oat nursery and a small trial using both this freshly dug and the powder-dry original soil was performed. Pots filled with these two soils were seeded and incubated out-of-doors from October 12 until November 19 when they were brought into the greenhouse. Mosaic developed in plants growing in either soil.

Experiment 4: On December 8, 38 pots were filled with the original soil, which was then brought to optimum moisture content; half were steamed at 8 psi for 2 hours., then all were seeded to Black Winter Oat, C. I. 4108, and incubated in the greenhouse maintained at 60° to 70° F. Mosaic first appeared in mid-March 1961, approximately 100 days after seeding. The day-length in the greenhouse was regulated at 12 hours. In this experiment with soil stored dry for about 4 months and with no outdoor incubation, plants in 5 of 19 unsteamed pots developed no mosaic.

No mosaic developed in plants grown in the steamed soil. The seed throughout was from diseased plants, so the absence of mosaic in the steamed series is evidence that the causal entity is either not seed-borne or very rarely so. The infectious property in the soil was not lost by drying for periods much in excess of 1 week at 20° C.

The recent expansion of knowledge of soil-borne viruses in England and Scotland, with the beet ringspot strain of tomato black ring virus (6, 7) and the tobacco rattle virus (2), both of which infect oats, as well as many other plants, led to inoculation trials to explore other possible affinities.

HOST RANGE STUDIES; MECHANICAL INOCULATIONS

On several different dates in diverse individual trials leaves of mosaic-infected oats showing good symptoms in the greenhouse experiments were cut into short pieces and macerated with mortar and pestle and the juice squeezed from the pulp through a moistened clean piece of cheese cloth. Mixed carborundum sizes (600 mesh and Ca. 340) kindly supplied by Mr. H. H. McKinney and also fine mesh carborundum (600) were used as abrasives with the juice in the several trials. The inoculum was applied to all test plants by gently rubbing the juice-carborundum mixture onto leaf surfaces between two fingers. The following plant species inoculated (other than Avena sativa) on which no visible symptoms developed are presumed negative:

CHENOPODIACEAE -- Beta vulgaris, Detroit dark red table beet, sugar beet; Spinacia oleracea, Improved thick leaf spinach.

COMPOSITAE -- Lactuca sativa, Black-seeded Simpson lettuce; Tagetes erecta, Sunset Giant marigold.

CRUCIFERAE -- Brassica rapa, Purple top white globe turnip; B. campestris var. napobrassica, American purple top rutabaga; Raphanus sativus, common radish.

CUCURBITACEAE -- Cucumis sativus, Boston Pickling, National Pickling cucumber; Cucurbita maxima, Hubbard squash; Curcurbita pepo, Sugar or New England Pie Pumpkin.

GRAMINEAE -- Bromus commutatus, J-42; B. inermis, Manchar; B. mollis, F-194; B. racemosus, J-41; B. secalinus, J-59; B. tectorum, J-15; Dactylis glomerata, Latar, Potomac, P-8775; Festuca arundinacea, Alta; F. elatior, S-215, P-3161; Hordeum leporinum, F-464; Lolium multiflorum; L. perenne, P-312; L. remotum; L. temulentum, 218-085; Phleum pratense, Engmo; Secale cereale, Sill's rye (13); Triticum aestivum, Michigan Amber; T. spelta, Red Winter; Zea mays, Improved Golden Bantam, Span Cross Yellow Hybrid.

LEGUMINOSAE -- Phaseolus vulgaris, Dwarf horticultural shell bean, Red Mexican U. I. 35, Pinto, Bountiful, Keeney Refugee; Pisum sativum, Perfected Wales pea; Vicia faba, broadbean; Vigna sinensis, cowpea.

ROSACEAE -- Fragaria chiloensis var. ananassa, Alpine strawberry.

SOLANACEAE -- Capsicum frutescens, California Wonder pepper; Lycopersicon esculentum, Washington State Forcing, Yellow Plum tomato; Nicotiana tabacum, Havana 38, 402 flue cured tobacco; Petunia hybrida, Large flowered mixed petunia.

TROPAEOLACEAE -- Tropaeolum majus, nasturtium.

UMBELLIFERAE -- Daucus carota, Danvers half long carrot; Pastinaca sativa, Hollow crown parsnip.

Special attention was given to Lolium spp. because of the Lolium-Dactylis mosaic virus of this area (1, 14) which infected oats in inoculation trials by mechanical means. No mosaic symptoms were found in Lolium in the fields where the oats became diseased.

The mosaic was transmitted only to oats in these tests and to it sporadically. In some trials no oats were diseased; in others, as many as 10 to 20% of the plants developed mosaic in from 8 to 15 days after inoculation. The variety Letoria was most consistently infected. The oat variety Statesville was suggested by McKinney as one of the varieties of oats most easily inoculated by mechanical means with the soil-borne mosaic of the Southeast. In one trial involving 60 seedlings each of Statesville and Letoria, several Letoria plants developed mosaic but none of the Statesville plants did. Statesville was not tested further.

DISCUSSION

The observations reported here do not permit precise determination of the relation of the Western Washington soil-borne oat mosaic virus with other viruses attacking oats. The data so far obtained show several points of similarity with the soil-borne oat mosaic viruses of southeastern United States, and no significant discrepancies. Neither is seed-borne, both persist for considerable periods in dry soil, both are essentially limited in natural occurrence to fall-seeded oats (a single plant of a spring oat, C. I. 4207, seeded in early April 1961 was mosaiced on July 7 at Vancouver, Washington), both are transmitted erratically and with difficulty by carborundum and expressed juice, and both have a prolonged "incubation" period in natural infections.

The failure to produce visible symptoms on dicotyledonous plants, even though the numbers of each species tested were small, and the persistence of the virus in dry soil seems to preclude close affinity with the soil-borne viruses of Great Britain that go to oats.

Linford and McKinney (9) found the parasitic myxomycete Polymyxa graminis Ledingham in roots of winter wheat suffering from soil-borne wheat mosaic. The association of the fungus with mosaic diseased wheat was consistent at several localities. The soil-borne wheat mosaic virus persists in dried soil (11), as does P. graminis (8), and both develop well at about 60° to 65° F. P. graminis was present in the roots of mosaiced oats screened from the dry soil in August 1960. This is apparently the first observation of P. graminis in oat roots. This occurrence in roots of oats affected by mosaic makes it particularly interesting. Another obligate parasite, Olpidium brassicae (Wor.) Dang., which has been associated with lettuce big vein (4), was also present in the oat roots. Nematodes are being increasingly incriminated as vectors of some soil-borne viruses. No experiments have been conducted as yet on the possible role of these varied organisms in the vection of the Western Washington soil-borne oat mosaic virus.

The soil-borne oat mosaic virus did not produce symptoms in Lolium spp. or in Dactylis glomerata, differentiating it from the mosaic of these hosts (1, 14) found in Western Washington. The Lolium-Dactylis mosaic produces similar symptoms on oats, however, following inoculation by rubbing.

Fall-seeded oats in this region are generally seriously affected by yellow dwarf (BYD), making observation of the mosaic difficult. Mosaic has been observed at other locations west of the Cascade Mountains, but is believed of minor significance in comparison to yellow dwarf which attacks all oats, both fall and spring planted, as well as wheat, barley, and several grasses.

Literature Cited

1. BRUEHL, G. W., H. TOKO, and H. H. McKINNEY. 1957. Mosaics of Italian ryegrass and orchard grass in Western Washington. (Abst.) Phytopathology 47: 517.
2. CADMAN, C. H., and B. D. HARRISON. 1959. Studies on the properties of soil-borne viruses of the tobacco-rattle type occurring in Scotland. Ann. Appl. Biol. 47: 542-556.
3. CADMAN, C. H., and B. D. HARRISON. 1960. Studies on the behavior in soils of tomato black ring, raspberry ringspot, and arabis mosaic viruses. Virology 10: 1-20.
4. GROGAN, R. G., F. W. ZINK, Wm. B. HEWITT, and K. A. KIMBLE. 1958. The association of Olpidium with the big-vein disease of lettuce. Phytopathology 48: 292-297.
5. HADDEN, S. J., and H. F. HARRISON. 1955. Occurrence of oat mosaic in the lower coastal plain of South Carolina. Plant Disease Reptr. 39: 628-632.
6. HARRISON, B. D. 1955. Relationship between beet ringspot, potato bouquet and tomato black ring viruses. J. Gen. Microbiology 18: 450-460.
7. HARRISON, B. D. 1957. Studies on the host range, properties, and mode of transmission of beet ringspot virus. Ann. Appl. Biol. 45: 462-472.

8. LEDINGHAM, G. A. 1939. Studies on Polymyxa graminis, n. gen. n. sp., A plasmodiophoraceous root parasite of wheat. Can. J. Research 17: 38-51.
9. LINFORD, M. B., and H. H. McKINNEY. 1954. Occurrence of Polymyxa graminis in roots of small grains in the United States. Plant Disease Reptr. 38: 711-713.
10. McKINNEY, H. H. 1946. Mosaics of winter oats induced by soil-borne viruses. Phytopathology 36: 359-369.
11. McKINNEY, H. H., W. R. PADEN, and BENJAMIN KOEHLER. 1957. Studies on chemical control and overseasoning of, and natural inoculation with, the soil-borne viruses of wheat and oats. Plant Disease Reptr. 41: 256-266.
12. McKINNEY, H. H., T. R. STANTON, J. L. SEAL, T. H. ROGERS, W. R. PADEN, G. K. MIDDLETON, and U. R. GORE. 1949. Mosaics of winter oats and their control in the southeastern states. U. S. Dept. of Agr. Circ. 809, 17 pp.
13. SILL, W. H., Jr. 1955. A winter rye useful in identifying soil-borne wheat mosaic virus. Phytopathology 45: 637.
14. SLYKHUIS, J. T. 1961. Differential hosts and new host records for five grass viruses. Can. J. Plant Sci. 41: 211-216.

DEPARTMENT OF PLANT PATHOLOGY, WASHINGTON STATE UNIVERSITY, PULLMAN, WASHINGTON

PINUS RADIATA SUSCEPTIBLE TO PITCH CANKER

George H. Hepting[1]

The pitch canker disease, caused by Fusarium lateritium pini Hepting, is common throughout the South on Virginia pine (Pinus virginiana), slash pine (P. elliottii), and longleaf pine (P. palustris). The fungus has also been demonstrated to be capable of killing many other species of pine (3, 4). In nature the pathogen usually causes only mortality of twigs and leaders, but with Virginia pine it frequently can girdle and kill a tree of any size. A copious flow of gum at the canker is its most obvious symptom (2).

Since Pinus radiata is planted in pure stands on a large scale in several countries, an experiment was carried out to test the pathogenicity of the pitch canker fungus to this species. Twenty-seven 1-0 seedlings of P. radiata, obtained from the Pacific Southwest Forest Experiment Station, were set out in three flats (nine to a flat) in a greenhouse at Asheville, North Carolina. Their average height was approximately 14 inches, with diameters averaging about 1/4 inch, 6 inches above the ground.

All seedlings promptly went into active shoot growth. On May 25, while actively growing, the nine seedlings in one flat were inoculated with a fresh isolate of the pitch canker Fusarium. Slits were made to the cambium and the bark sprung slightly. A piece of agar from an active culture was inserted. The incisions were covered with paper. The nine seedlings in another flat were inoculated with a year-old isolate of this fungus, and the nine in the third flat received the same bark slits made in the inoculated trees, but instead of inserting a bit of agar cut from an active culture, sterile agar was used.

Additional inoculations were made on nine shoots of P. virginiana, a very susceptible host of the pitch canker Fusarium. Four check incisions with plain agar were also made on this species.

After 3 weeks all inoculations on both tree species, with both the fresh and the year-old isolate, had taken. Sunken lesions had developed with much pitch flow from P. virginiana, and sunken lesions with very slight pitch flow from P. radiata. None of the 13 check incisions developed lesions.

Eight weeks after inoculation the lesions had girdled the stems; most of the Virginia pine shoots were already dead; and all of the P. radiata trees had wilted, faded, and were dying (Fig. 1). At this time pink sporodochia of the pathogen were observed breaking through the bark near the lesions, mostly from lenticels. They yielded abundant macroconidia which, upon culturing, produced colonies of F. lateritium pini identical with those used in the inoculations. Isolations from the lesion margins also yielded the same fungus.

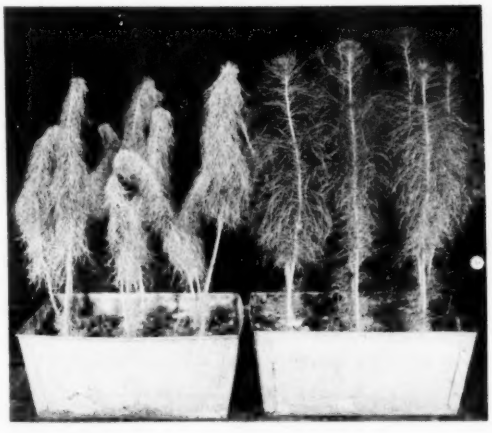


FIGURE 1. Left, a flat of P. radiata seedlings dying from girdling lesions following inoculation with the pitch canker fungus. Right, a flat of seedlings receiving plain agar in incisions. These remained healthy.

[1]Chief, Division of Forest Disease Research, Southeastern Forest Experiment Station, United States Department of Agriculture, Forest Service, Asheville, North Carolina.

This experiment demonstrated that P. radiata in the seedling stage is highly susceptible to F. lateritium pini, and that, in view of the capacity of this fungus to kill twigs, seedlings, and larger trees of many pine species in the southern United States, as well as Scots pine planted there, this fungus must be considered a potential danger to P. radiata wherever it is grown. To realize this potential the fungus first must reach concentrations of P. radiata, and then have the capacity to spread. This pathogen seems to be a wound parasite and any buildup would likely be contingent on an effective vector or injuries suitable for infection.

Diligent searches for a fruiting stage of the pitch canker fungus in nature in the United States have previously been almost wholly unsuccessful, except on the surface of fresh wounds sprayed with a spore suspension (1). Hence it is significant that abundant normal sporodochia formed on the induced lesions on P. radiata even before the seedlings were dead.

Literature Cited

1. BERRY, CHARLES R., and GEORGE H. HEPTING. 1959. Pitch canker of southern pines. U. S. Dept. of Agr. Forest Pest Leaflet 35, 3 pp.
2. CLAPPER, RUSSELL B. 1954. Stimulation of pine oleoresin flow by fungus inoculation. Econ. Botany 8: 269-284.
3. HEPTING, GEORGE H., and ELMER R. ROTH. 1946. Pitch canker, a new disease of some southern pines. J. Forestry 44: 742-744.
4. HEPTING, G. H., and E. R. ROTH. 1953. Host relations and spread of the pine pitch canker disease. (Abst.) Phytopathology 43: 475.

SOUTHEASTERN FOREST EXPERIMENT STATION, FOREST SERVICE,
UNITED STATES DEPARTMENT OF AGRICULTURE, ASHEVILLE, NORTH CAROLINA

SOME EVIDENCE THAT PEAR BARK MEASLES IS SEED-BORNE

C. B. Cordy[1] and I. C. MacSwan[2]

The name pear bark measles has been applied to a virus-like malady of pears in California. California workers report that symptoms have been seen on the Bartlett, Bosc, Comice, D'Anjou, Farmingdale, Hardy, Lemon, Max Red Bartlett and Winter Nellis varieties[3]. Surveys of Medford, Oregon pear orchards have shown bark measles to be present on a few trees of the Bartlett varieties with the following rootstock or rootstock-interstock combinations.

Variety	Interstock	Rootstock
Bartlett	None	French Seedling
Bartlett	Old Home	Calleryana Seedling
Bartlett	Old Home	French Seedling
Red Bartlett	Bosc	French Seedling
Red Bartlett	D'Anjou	French Seedling
Red Bartlett	Winter Nellis	French Seedling

The use of buds or grafting wood from affected trees has been considered the principal means of spreading bark measles. Distribution of bark measles by other means is not known. Our test with seeds from affected trees indicates that bark measles may be seed-borne.

Seeds from open-pollinated fruits of Red Bartlett trees growing near numerous Packham's Triumph trees which could have served as the male parent were gathered in the Medford area in September 1956. These seeds were planted in soil in plant bands and kept at room temperature. The seedlings were transplanted to ground beds in March 1957. Pear bark measles symptoms have been noticeable in some of the seedlings since 1958. By August 1961, sixteen of the forty-five seedlings were showing typical symptoms and in some cases symptoms appeared even on the current season's growth. Symptoms of measles are shown in Figure 1.

While the seed planting did not involve best isolation techniques, this initial test indicates that pear bark measles may be seed-borne; therefore, pear seed as well as buds and grafting wood should be selected only from trees free of the measles symptoms.

FIGURE 1. Symptoms of pear bark measles on: A -- 5-year-old pear seedling. B -- Bartlett.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY AND FEDERAL COOPERATIVE EXTENSION SERVICE, OREGON STATE UNIVERSITY, CORVALLIS, OREGON

[1]County Extension Agent, Oregon State University, Federal Cooperative Extension Service, Medford, Oregon.

[2]Plant Pathology Specialist, Oregon State University, Federal Cooperative Extension Service, Corvallis, Oregon.

[3]Millecan, A. A., Carl W. Nichols and William M. Brown, Jr. 1961. Pear bark measles and its association in California with old home interstocks. (Abstracts of papers presented at the Forty-third Annual Meeting, Pacific Division of the American Phytopathological Society, Davis, California.)

THE EFFECT OF PESTICIDES ON TURFGRASS DISEASE INCIDENCE[1]

John H. Madison[2]

Abstract

Insecticides, fungicides, herbicides and a nematocide were applied to a young Kentucky bluegrass turf in June. Shortly thereafter, a severe and uniform attack of Rhizoctonia solani Kuehn occurred and was evaluated by string transect. Insecticides did not affect disease incidence; fungicides gave a non-significant decrease; herbicides increased disease incidence and two, calcium arsenate and a phenyl complex, increased disease significantly; the nematocide significantly decreased disease severity and as injurious nematodes were not present, the material is considered to affect the disease organism.

INTRODUCTION

Letters from homeowners indicate that turfgrass disease may be promoted by commercial pesticides or by the manner in which they are used. The public often misuses such materials, but life forms are interrelated, and materials severely injurious to one organism may well affect another. This possibility was investigated, with respect to turfgrasses, by examining the susceptibility to infection of turf treated with representative fungicides, herbicides, insecticides, and a nematocide.

METHODS AND MATERIALS

Test plants were mature Kentucky bluegrass turf and a new stand of bluegrass turf (sown February 24, 1960). The test chemicals were proprietary formulations and the active ingredients are listed in Table 1. On mature bluegrass, applications were made at the rates and seasons recommended by the manufacturers. The new seeding of bluegrass was not ready for use at the earlier dates, so all materials, including pre-emergence herbicides, were applied once, June 16.

An unsuccessful attempt was made to induce Helminthosporium in the foot-rot stage by lowering the 1 1/4-inch height of cut to 7/8 inch, to cause stress, and by spraying the plots with a slurry of a locally pathogenic isolate of Helminthosporium vagans Drechs. grown on agar. The plots were inoculated in the evening, and were then wetted and covered with a polyethylene tarpaulin from 7:00 p.m. to 7:30 a.m., on that and four succeeding nights. The mature stand appeared disease-free throughout the summer, but the plot of new Kentucky bluegrass, inoculated on June 26, showed symptoms of Rhizoctonia solani Kuehn shortly thereafter, and a uniform and severe infection of brown-patch developed. The area had been continuously in barley for several years previously, which may account for the presence of inoculum. The infection, judged to be at its peak in the third week of July, was evaluated then, once by the author and 1 week later by an assistant, each counting in different directions. Evaluation was made by a string transect method. Five strings, 60 inches long, were stretched 6 inches apart across a steel frame. The frame was dropped on a plot and the number of inches of string in contact with diseased areas of turf was counted. The mean of the two counts provides the data used. Individual plots were 5 x 6.5 feet. Thirteen treatments were replicated four times in an incomplete Latin-square design obtained by randomizing plan 13.5 in Cochran and Cox[3].

RESULTS AND DISCUSSION

Data were analyzed for recovery of inter-block information. Adjusted treatment means are given in Table 1. Differences were highly significant. Rhizoctonia was significantly more

[1]Lawrence J. Petersen, Department of Plant Pathology, University of California, Davis prepared the Helminthosporium inoculum and isolated and identified the Rhizoctonia solani. Armand R. Maggenti, Department of Nematology, UCD, identified the nematodes. Tokuwo Kono assisted with the transect counts. Their assistance is acknowledged with gratitude.

[2]Department of Landscape Horticulture, University of California, Davis, California.

[3]Cochran, W. G., and Gertrude M. Cox. 1957. Experimental Designs. John Wiley & Sons, Inc., N. Y. pp. 509, 523.

Table 1. Effects of pesticides on the severity of Rhizoctonia solani on Kentucky bluegrass (Poa pratensis.)

Material	Rate of application per 1000 sq. ft.	Adjusted mean (inches dead of 300-inch transect)
1. Control		69.2
Insecticides		
2. DDT - 50% wettable powder	2.5 lb	69.4
3. 4% dieldrin + 1% lindane on granular carrier (Ortho Lawn Moth Control)	1.43 lb	64.1
Mean		66.8
Fungicides		
4. Captan	0.2 lb	59.2
5. Phenylmercuric acetate, 10% solution (PMAS)	1.0 oz	61.2
Mean		60.2
Pre-emergence herbicides		
6. Tricalcium arsenate 73% granular (Chip-Cal)	12.0 lb	93.9
7. Technical chlordane-23% on vermiculite (Halts)	6.0 lb	60.2
8. O-(2,4-dichlorophenyl) O-methyl isopropylphosphoramidothioate (Zytron)	5. lb	124.3
Mean		92.8
Selective herbicides		
9. Disodium methyl arsenate 18-9% solution (Di-Met)	94.6 ml	77.2
10. 2,4,5-trichlorophenoxypropionic ester - 4 lbs/gal (Kuron)	32.6 ml	82.7
11. 2,4-D ester 13.8% - 2,4,5T ester 6.6% - (Weed-B-Gon)	120. ml	62.5
Mean		74.1
Nematocide		
12. 1,2-dibromo-3-chloropropane 67% - solution (Nemagon)	173.7 ml	49.6
13. Mixture - 2 + 3 + 5 + 9		64.0

Means significantly different from the control (5% level) are those ≥ 85.8 or ≤ 52.7.

severe (5% level) on plots receiving calcium arsenate and the phenyl complex, and less severe on those receiving the nematocide. Results were consistent with appearance in the field. On both young and mature bluegrass, the phenyl complex suppressed growth, so less grass was removed from those plots by mowing. Plots receiving the phenyl complex or calcium arsenate were visibly paler, a difference that persisted 2 months or longer. Transient visible injury to turf was also caused by 2,4,5-TP and disodium methylarsenate, but recovery was rapid. Incidence of Rhizoctonia on these plots was greater than the values clustered around the control, but failed of significance. Nonetheless, the four high values are of interest because they are all from plots receiving herbicides. The insecticides and fungicides tested appear to perform their function without affecting the bluegrass. Chemicals used to poison other species of flowering plants, however, may seriously affect bluegrass vigor as measured by disease resistance.

Results from the nematocide were of interest. Several attempts to study nematodes on this area have failed because the numbers are too few for results. Most nematode counts on an adjacent area yielded fewer than 10 Pratylenchus thornei per 100-gram soil sample. One must assume, then, that benefits from the nematocide are a direct effect on Rhizoctonia rather than an indirect effect from nematode control.

Calcium arsenate and the phenyl complex, both pre-emergence herbicides, would normally be applied earlier in the season when temperatures are cooler and more favorable for growth of grass. Thus, the experiment is in part a test of misuse of the products. Even so, properly timed applications (March) visibly injured the mature grass, indicating that the results correctly show injurious effects from the materials, though the magnitude of the injury may be less when applied in season.

The fact that two pre-emergence herbicides favored the incidence of Rhizoctonia brownpatch disease on turf may suggest to some a new parameter for testing materials for crop tolerance.

DEPARTMENT OF LANDSCAPE HORTICULTURE, UNIVERSITY OF CALIFORNIA, DAVIS

CEREAL RUST EPIDEMIOLOGY IN KANSAS IN 1959[1]

L. E. Browder, C. O. Johnston, and S. M. Pady[2]

Summary

Abundant overwintering of severe fall infections of wheat leaf rust in most areas of Kansas in 1958-59, combined with a cool moist growing season, resulted in a heavy epidemic of that disease, causing a loss estimated at 10%. Wheat stem rust development was late and light except in the northeastern area of Kansas and damage from it was only a trace. The oat and barley rusts did not develop appreciably and did no damage. Stripe rust of wheat was not observed in 1959 in Kansas.

This paper is a continuation of a series of Kansas cereal rust epidemiology reports (1, 2, 3, 4, 5, 6, 7) begun in 1954. It is believed that such reports on a cumulative basis will provide valuable information for interpretation of future epidemiology work and for possible forecasting attempts.

The epidemiology of cereal rusts in Kansas in 1959 was studied by three methods, as follows: 1) gravity spore traps, 2) examination of rust susceptible wheat samples received from five branch experiment stations weekly, and 3) extensive survey trips. In addition to other climatic data that were available, data on the occurrence of dews were obtained through the use of a Taylor dewmeter[3] operated during the rust season. A rather detailed account of cereal rust development in Kansas in 1958-59 was prepared from these sources of information.

The outstanding feature of the 1958-59 season was the epiphytotic of wheat leaf rust, Puccinia recondita Rob. ex Desm., which resulted in a 10% loss, the second highest on record. Favorable weather conditions not only resulted in a wheat crop for the State of 209,700,000 bushels, but also favored a heavy fall buildup of leaf rust. During the winter, conditions were favorable for leaf rust and overwintering occurred in many areas throughout the State. Leaf rust thus was present in the fields much earlier than normal and developed rapidly. Leaf rust of rye also overwintered but other cereal rusts did not. They appeared late and did not develop sufficiently to cause damage.

FALL AND WINTER WEATHER, CROP, AND RUST CONDITIONS

A relatively cool, wet August favored growth of volunteer grain in most portions of Kansas in 1958. By September 9, both leaf rust and stem rust were present on volunteer wheat, oats and barley at Manhattan, abundant leaf rust was found on a wheat sample sent in from Colby on this date. A survey trip to central Kansas, September 22, revealed rapid development of leaf and stem rust on volunteer wheat; seeding operations were in progress at that time. Ideal seeding and emergence weather made possible a rapid emergence of the wheat crop over the State. Inoculum from remaining rusted volunteer was available for infections of the seeded crop during October. Temperatures remained sufficiently warm for continued rust development into November and wetting periods from dews were frequent.

Winter temperatures were generally mild; however, some extremely low temperatures did occur, notably a -23° F at Manhattan, January 3, as well as comparable temperatures over the State. However, snow cover was present in most areas where low temperatures occurred, which apparently gave a large measure of protection to both the wheat crop and overwintering rust infections.

---

[1]Contribution No. 584, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan. Botany serial No. 745.

[2]Plant pathologist and pathologist, Crop Research Division, Agricultural Research Service, United States Department of Agriculture; and Head of Department of Botany and Plant Pathology, Kansas State University, Manhattan, respectively.

[3]Patterned after Taylor, C. F. 1956. A device for recording the duration of dew deposits. Plant Disease Reptr. 40:1025-1028.

OVERWINTERING OF WHEAT LEAF RUST

One of the most important factors affecting the development of the severe wheat leaf rust epidemic was abundant overwintering as dormant mycelium in most areas of the State.

Overwintered pustules were first observed on a sample received from Elkhart, March 16, on which small primary pustules were present on the lower leaves. Additional evidence of overwintering was found on survey trips over the western, south-central, and southeastern parts of the State from March 30 to April 3. During that time, traces of leaf rust were observed in most fields examined. On April 6, examination of many fields in central Kansas revealed scattered leaf rust pustules in a few fields. At that time, wheat streak mosaic was killing the lower leaves in most fields in that area. It is believed that the abundance of this disease was important in lowering the severity of overwintering leaf rust in central and western Kansas. In other areas of the State, leaf rust of wheat continued to develop slowly throughout a cool April.

Overwintering of leaf rust was particularly abundant in southeastern, south-central, and some localities of north-central Kansas. On April 8, spreader rows of Columbia wheat in a leaf rust variety test nursery at Ashland farm near Manhattan were recorded as having a 5% severity of overwintered pustules on the lower leaves, an extremely heavy severity of overwintering pustules. Commercial fields in the same locality were erratic in the amount of overwintered leaf rust, probably as a result of seeding date and proximity to fall inoculum from volunteer wheat. On April 13, only 5 days later, leaf rust was developing perceptibly from the overwintered inoculum in the Manhattan area.

An important feature following overwintering of wheat leaf rust in many fields was an apparent disappearance of the rust because powdery mildew, Erysiphe graminis DC., killed the lower leaves. This disappearance was only temporary, however, as leaf rust soon reappeared on higher leaves in even greater amounts.

Samples of susceptible wheat plants were first received at Manhattan from five branch experiment stations in Kansas, April 22. No rust was found on the samples from Hays and Colby, traces of leaf rust were found on the Hutchinson and Garden City samples, and leaf rust was recorded at 5% severity and 75% prevalence at Mound Valley in southeastern Kansas (Table 1).

Table 1. Severity and prevalence of leaf rust of wheat at six locations in Kansas in 1959, based on samples received from field stations.

Date	Plant areas infected	Geographic part of the State and the station of collection					
		S. E.	Central	S. W.	N. W.	N. C.	N. E.
		Mound Valley	Hutchinson	Garden City	Colby	Hays	Manhattan
April 1		T(O W)[a]	T(O W)	T(O W)	T(O W)	T(O W)	T(O W)
22		5/75[b]	T/T	0	0	0	9 pust/100 tillers
29		5/75	1/60	0	0	0	
May 6		40/100	5/75	T/T	0	0	T/T
13	mid. leaves	70/100	50/100	T/T	1 pust/8-10 plants	T/T	20/60
	flag leaves	20/100	2/50	--			5/75
12[22]	lower leaves	dead	dead	T/100	T/100	T/100	20/100
	flag leaves	80/100	20/100				T/100
27	flag leaves	70/100	60/100	T/100	T/100	1/100	50/100
June 3	flag leaves	dead	70/100	70/100	30/100	50/100	90/100

[a](O W) = overwintered; T= Trace.

[b]% severity/% prevalence.

Leaf rust was common at that time at Manhattan, averaging about 2% severity on the lower leaves. On the same date, a survey trip to south-central Kansas counties revealed up to 5% severity and 75% prevalence in many fields in Marion, Harvey, and McPherson counties. These severities occurred about the time leaf rust is found in most years as traces of scattered primary pustules resulting from airborne inculum (4). The importance of overwintering in the 1959 leaf rust epidemic is evident from these data. The amount of leaf rust overwintering was the greatest ever in many years of observation of the cereal rusts in Kansas.

SPRING DEVELOPMENT OF WHEAT LEAF RUST

Weather conditions were generally favorable for buildup of wheat leaf rust from the overwintered infections. On April 29, severities of 20 to 40% on middle leaves and 5% on flag leaves were observed generally in south-central Kansas. By the first week in May, such infections were general throughout the eastern two-thirds of the State as indicated by samples received from Mound Valley and Hutchinson (Table 1). Leaf rust had caused premature defoliation of much of the wheat as far west as Hays by May 20, although heavy wheat streak mosaic virus infections tended to confuse the situation in some of the central portion. Leaf rust developed heavily in southwestern Kansas late in the season (Table 1), whereas relatively light infections occurred in the northwestern district in the vicinity of Colby, apparently from lack of sufficient wetting periods.

Temperature, wetting period, rainfall, wind, and spore trap data from Manhattan are presented in Figure 1. Particularly important, as shown from these data, was the occurrence of an infection period on each of 18 consecutive nights from May 1 to 18, perhaps the most critical period in rust buildup in Kansas. Temperatures were also favorable for leaf rust development through most of April and May.

Silicone-coated microscope slides were exposed daily in a gravity spore trap at the Agronomy farm northwest of the Kansas State University campus at Manhattan, from April 17 to June 25. Leaf rust spores were first trapped April 25, after overwintered rust had been

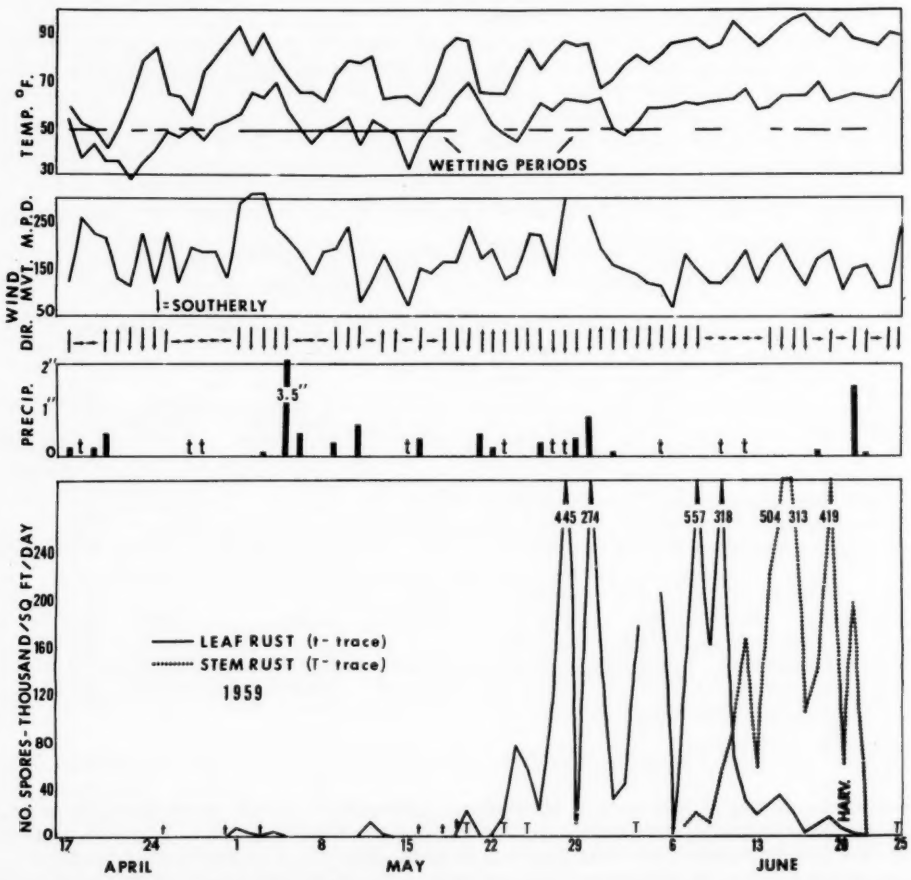


FIGURE 1. Spore numbers trapped and meteorological data for April 17 through June 25, 1959 at Manhattan, Kansas. Spore trap exposed at Agronomy Farm, Kansas State University; meteorological data from Agronomy Farm Weather Station.

observed in quantity in local fields. Relatively small numbers of leaf rust spores were caught from April 30 to May 4 (Fig. 1) when southerly winds occurred. The exact source of these airborne spores is not known since a great buildup of overwintered infections had already occurred in many sections of the State. Airborne spores, along with spores disseminated locally from overwintered rust, favored by near-optimum environmental conditions, resulted in a so-called rust "explosion" during the last half of May. This caused rather severe damage to the wheat crop and an estimated loss in yield of 10%.

The explosive increase in leaf rust is evidenced by the tremendous number of spores trapped from May 20 to June 20 at Manhattan. A peak of 557,000 spores/square foot per day was reached June 8. A lesser peak of 445,000 spores/square foot per day was caught May 28. The number of leaf rust spores trapped subsided rapidly after about June 10.

A definite and exacting separation of the importance of overwintered and airborne inoculum from Oklahoma and Texas was difficult; however, overwintered rust was certainly important in the very early inception of the 1959 leaf rust epidemic. Though some details of the development of leaf rust in 1959 are not fully clear, four facts remain prominent: 1) leaf rust of wheat overwintered in unusual abundance, 2) it developed to epidemic proportions unusually early in the eastern two-thirds of Kansas, 3) it caused serious losses in many fields and many parts of the State, and 4) tremendous potential inoculum was produced to be transported northward.

WHEAT STEM RUST

Airborne stem rust spores were first caught on slides at Manhattan on May 20 (Fig. 1) and this rust, Puccinia graminis Pers. f. sp. tritici Eriks. & E. Henn., was first found on wheat in trace amounts May 28, both in south-central Kansas and in the vicinity of Manhattan. This is about average for stem rust, which usually appears in Kansas around June 1 (4). Only small numbers of spores were observed until June 6 when second-generation pustules from local infections matured. Severities in focal fields, as well as spore numbers caught, increased very rapidly in central and northeastern Kansas in the ensuing 14 days. A peak of 504,000 spores/square foot was observed June 15. The buildup of wheat stem rust came quite late in the crop development period and therefore did little damage except in the northeastern area where some measurable damage occurred. By June 20, harvest is usually underway in southern Kansas and moves northward steadily, reaching northern Kansas about July 4. The heavy spore showers of June 15 thus arrived too late to injure the crop and the over-all loss from stem rust was estimated at only a trace.

STRIPE RUST

No stripe rust, Puccinia striiformis West., infections were found in Kansas though careful search was made throughout the rust development season. Although this disease developed in trace amounts in northern Texas, drouth and high temperatures apparently stopped its development there. Stripe rust was not reported north of Texas in 1959; its absence in Kansas in 1959 makes its occurrence in 1957 (7) and damage in 1958 (6) seem even more unusual than previously believed.

OTHER CEREAL RUSTS

Leaf rust of rye, P. recondita Rob. ex. Desm. (P. dispersa Eriks. & E. Henn.), overwintered as abundantly as wheat leaf rust and its development closely parallels that of wheat leaf rust. However, the small acreage of rye in Kansas leaves the damage caused by rye leaf rust of little economic significance.

Oat crown rust, P. coronata Cda., and oat stem rust, P. graminis Pers. f. sp. avenae Eriks. & E. Henn., were observed in only trace amounts and caused no measurable losses. The oat redleaf (barley yellow dwarf) virus defoliated much of the oat crop in Kansas and left little tissue for rust development. Occasional light infections of crown and stem rust were noted on Kanota which appeared resistant to the red leaf virus.

Literature Cited

1. PADY, S. M., and C. O. JOHNSTON. 1955. The concentration of airborne rust spores in relation to epidemiology of wheat rusts in Kansas in 1954. Plant Disease Reptr. 39:463-466.
2. PADY, S. M., and C. O. JOHNSTON. 1956. Aerobiology of rust spores and epidemiology of wheat rusts in Kansas in 1955. Plant Disease Reptr. 40:882-885.
3. PADY, S. M., and C. O. JOHNSTON. 1956. Cereal rust epidemiology and aerobiology in Kansas in 1956. Plant Disease Reptr. 40:1061-1064.
4. PADY, S. M., and C. O. JOHNSTON. 1958. Cereal rust epidemiology and aerobiology in Kansas in 1957. Plant Disease Reptr. 42:726-733.
5. PADY, S. M., and C. O. JOHNSTON. 1959. Stripe rust in Kansas in 1958. Plant Disease Reptr. 43:159-163.
6. PADY, S. M., and C. O. JOHNSTON. 1959. Cereal rust aerobiology and epidemiology in Kansas in 1958. Plant Disease Reptr. 43:607-612.
7. PADY, S. M., C. O. JOHNSTON, and C. T. ROGERSON. 1957. Stripe rust of wheat in Kansas in 1957. Plant Disease Reptr. 41:959-961.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, KANSAS STATE UNIVERSITY, MANHATTAN IN COOPERATION WITH CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

JUNCUS EFFUSUS, A NEW HOST FOR EPICHLOË TYPHINA[1]

R. A. Kilpatrick[2], Avery E. Rich[3], and J. G. Conklin[3]

Summary

Cat-tail disease caused by Epichloë typhina was found in June 1961 on Juncus effusus at Durham, New Hampshire. Larvae of an unidentified dipterous insect were associated with the fungus tissue. This is the first observation of the disease in New Hampshire and Juncus spp. are believed to be new hosts for the fungus.

Two diseases (2) have been reported on Juncus effusus, commonly known as bog rush. This report lists an additional disease, and it is believed to be the first report of Epichloë typhina (Pers. ex Fr.) Tul. on a member of the Juncaceae (rush family).

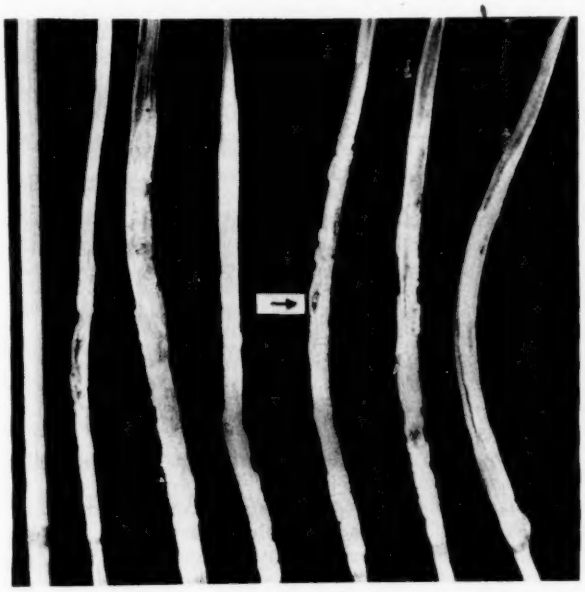


FIGURE 1. Epichloe head blight on stems of Juncus effusus. Left -- healthy stem. Right -- six stems with heavy infection. Note the larva embedded in the stromatic mass and girdling of fungus tissue by other larvae.

E. typhina, causal agent of the cat-tail disease (1) (Fig. 1), was found on stems of J. effusus in June 1961 at Durham, New Hampshire. Two areas of heavily infected plants were found in a ditch near a reservoir. Nearby Gramineae were free of infection.

In mid-June white mycelial strands were spreading over the nodes and internodes. Light-yellow to orange stromatic masses with visible perithecia were noted on stems with advanced infection. Seed development was prevented on diseased plants.

When the affected areas were revisited 4 weeks later diseased stems were gray and lacked fresh mycelial development. Spread of the disease had evidently been inhibited by high temperatures.

Unidentified dipterous larvae were embedded in the stromatic tissue and feeding on it. After extensive feeding, the stromata were girdled. Attempts to rear the insect are being made by members of the Department of Entomology at Durham.

This report is the first known occurrence of Epichloë typhina in New Hampshire. Specimens have been deposited with Dr. C. R. Benjamin, Mycology Investigations, Crops Protection Research Branch, Beltsville, Maryland.

Literature Cited

1. SPRAGUE, R. 1960. Diseases of cereals and grasses in North America, Ronald Press. New York. pp. 77-78.
2. UNITED STATES DEPARTMENT OF AGRICULTURE. 1960. Index of Plant Diseases in the United States. Agriculture Handbook No. 165.

DEPARTMENTS OF BOTANY AND ENTOMOLOGY, UNIVERSITY OF NEW HAMPSHIRE, DURHAM

[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and Departments of Botany and Entomology, New Hampshire Agricultural Experiment Station, Durham. Published with the approval of the Director of the New Hampshire Agricultural Experiment Station as Scientific Contribution No. 278.

[2]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.
[3]Plant Pathologist and Entomologist, respectively, New Hampshire Agricultural Experiment Station.

DUTCH ELM DISEASE IN ARKANSAS[1]

Charles L. Wilson, M. C. McDaniel, and C. P. Seymour[2]

Dutch elm disease has been found in four northeastern counties of Arkansas. This is the first confirmed report of the occurrence of this disease in Arkansas. Elm phloem necrosis was detected in the State in 1945[3].

A limited survey was made during July and August 1961 in the northeastern part of Arkansas and six trees were found that yielded cultures of Ceratocystis ulmi (Buis.) Moreau. Five of the trees were Ulmus americana and one located at Blytheville was U. alata. The original culture of C. ulmi was identified by Dr. A. F. Verrall, USFS, New Orleans.

Two positive Dutch elm diseased trees were found in Truman (Poinsett Co.), two in Pocahontas (Randolph Co.), one in Blytheville (Mississippi Co.), and one in Paragould (Greene Co.).

The Arkansas State Plant Board has had all the diseased trees burned. A co-operative program is being developed by the University of Arkansas, Arkansas State Plant Board, and the Arkansas State Forestry Commission to inform local officials of this problem and to formulate control practices. A more extensive survey will be made in 1962 for this disease.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF ARKANSAS, FAYETTEVILLE

[1]Published with the approval of the Director of the Arkansas Agricultural Experiment Station. Appreciation is expressed to Dr. A. F. Verrall for confirming the identification of C. ulmi and for his helpful suggestions.

[2]Assistant Professor, University of Arkansas; Extension Plant Pathologist, Agriculture Extension Service; Chief, Division Plant Pathology and Entomology, Arkansas State Plant Board, respectively.

[3]Larsh, H. W. 1945. Elm phloem necrosis in Arkansas and Oklahoma. Plant Disease Reptr. 29: 699-700.

HOJA BLANCA IN EL SALVADOR

Oscar Ancalmo and Guillermo Ramos Siliézar[1]

Hoja Blanca, a virus disease of rice (Oryza sativa), was recorded for the first time in El Salvador in 1958[2] on a farm near the Pacific Coast.

In 1959 a survey throughout the rice-growing areas of the country showed that the disease was generally established in fields along and near the south coast. Field reductions of as much as 80% in such varieties as Bluebonnet-50 and Toro were recorded. Other susceptible varieties such as Sunbonnet, Nira, Nirola, Rexoro, Century Patna 231 and some criollo varieties were observed to have the disease at the Santa Cruz Porrillo Experiment Station (2).

Large populations of the insect vector Sogata orizicola Muir. have been noted in all fields where the disease was observed. Other species of Sogatas present in the country are S. cubana Crawford and S. furcifera Horvath.

Field observations made in 1960 and 1961 showed that the incidence of disease has decreased in the country.

Gulf Rose (CI-9416), introduced to the country in 1959 because of its resistance to hoja blanca, was severely attacked in 1960 by the rice blast fungus Piricularia oryzae Cav. (1).

A cooperative project by the Dirección General de Investigaciones Agronómicas of El Salvador with the United States Department of Agriculture is in progress to find hoja blanca resistant material.

Literature Cited

1. ANCALMO, OSCAR, and ARMANDO ALAS LOPEZ. 1961. Rice blast in El Salvador. Plant Disease Reptr. 45: 576.
2. MOLINA, JOAQUÍN. 1960. Hoja Blanca. Agricultura en El Salvador 2: 13-15.

DIRECCIÓN GENERAL DE INVESTIGACIONES AGRONÓMICAS, SANTA TECLA, EL SALVADOR, C. A.

[1]Respectively, Chief Department of Plant Pathology and Entomologist, Entomology Department, Dirección General de Investigaciones Agronómicas, Santa Tecla, El Salvador.

[2]Acknowledgment is given to Bowen S. Crandall for identification of the disease.

A SEVERE EPIDEMIC OF HELMINTHOSPORIUM BROWN SPOT DISEASE ON CULTIVATED WILD RICE IN NORTHERN MINNESOTA

George A. Bean and Richard Schwartz[1]

Brown spot disease (Helminthosporium oryzae B. de Hann) was found in epidemic proportions on wild rice (Zizania aquatica) under commercial cultivation at the Chun King Corporation farm, Zim, Minnesota. The Chun King farm has at present more than 20 acres of wild rice under cultivation. Each year since the fields were established brown spot disease has increased in severity. In 1961, in one 10-acre field where wild rice had been grown for 3 consecutive years, there was a complete crop failure resulting from defoliation and lodging.

Disease severity appears related to the plant population because in younger fields nearby with thinner stands, brown spot was less severe. Drought during the 1961 growing season may also have been important, as Hemmi and Suzuki[2] found that cultivated rice (Oryza sativa) grown under dry conditions was more susceptible to attack by H. oryzae.

Attempts to control this disease are being made by application of fungicides both as dormant and foliage sprays, removal of crop debris, and the selection of resistant plants.

DEPARTMENT OF PLANT PATHOLOGY AND BOTANY,
INSTITUTE OF AGRICULTURE, UNIVERSITY OF MINNESOTA,
ST. PAUL, MINNESOTA

---

[1]Research Assistant, Department of Plant Pathology and Botany, University of Minnesota, St. Paul, Minnesota, and Agronomist, Chun King Corporation, Duluth, Minnesota, respectively. Experimental Paper No. 4716.

[2]Hemmi, T. and H. Suzuki. 1931. On the relation of soil moisture to the development of the Helminthosporium disease of rice seedlings. Forsch. Geb. PflKrankh., Kyoto, i, pp. 90-98. (Japanese, with English summary).